

Introduction to Anesthesia

THE PRINCIPLES OF SAFE PRACTICE

ROBERT D DRIPPS, M D Professor and Chairman, Department of Anesthesiology, Schools of Medicine, University of Pennsylvania, Anesthetist, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania

JAMES E ECKENHOFF, M D Professor of Anesthesiology, Schools of Medicine, University of Pennsylvania, Anesthetist, Hospital of the University of Pennsylvania, Philadelphia, Pennsylvania

LEROY D VANDAM, M D Clinical Professor of Anesthesia, Harvard Medical School, Director of Anesthesia, Peter Bent Brigham Hospital, Boston, Massachusetts

Line drawings by **LEROY D VANDAM, M D**

W B Saunders Company

1957 Philadelphia and London

© 1957, by W B Saunders Company

Copyright under the International Copyright Union

All Rights Reserved

This book is protected by copyright

No part of it may be duplicated or reproduced in any manner without written
permission from the publisher

Made in the United States of America Press of W B Saunders Company

LIBRARY OF CONGRESS CATALOG CARD NUMBER 57-12777

DEDICATION

To a surgeon who recognized the need for the development of anesthesiology, and contributed to its growth with vigor and enthusiasm — *Dr I S Razdin*

PREFACE

THIS BOOK is a descendant of a smaller work privately printed in 1949 and circulated in the Department of Anesthesiology of the Hospital of the University of Pennsylvania. It was called "Organization and Procedures." A second and larger edition appeared in 1953. The third lineal descendant is now being made available to others.

Much of the teaching of anesthesia is by word of mouth. Beginners seek to learn countless details that cannot be found in general texts. This is the lore of anesthesia that must be passed on from individual to individual. A good bit of the material gathered in this volume might be classified as being in this rather shadowy area, including such topics as the philosophy of records, surgeon-anesthetist relations, the value of death reports, hazards of the immediate postoperative period, the treatment of immediate postoperative pain and excitement, and the determination of the depth of general anesthesia.

The subjects and the manner of their presentation represent the thinking and ultimate distillation into teaching practice of the senior staff at Pennsylvania augmented and refined by all who came within teaching range. One who contributed much to the early editions and whose influence may be discerned in the present volume is Austin Lamont. We mention him with respect and give thanks to others who will find bits of their practice and philosophy in print.

Chapters dealing with fundamental aspects of certain techniques and with basic considerations of the drugs used in anesthesia have been included in the hope that such material can contribute to the safer practice of anesthesia. These are guides, they contain what we believe to be established precepts, but they are not provided in detail nor can they be regarded as complete.

We have intentionally omitted discussion of most of the specialized aspects of anesthesia such as hypothermia, deliberate hypotension,

hypnosis, the technical considerations of regional anesthesia and problems of the treatment of pain. These comprise advanced study in the field. We believe that the student for whom this book is intended should not be confronted by them until later in his training.

We trust that this book will be instructive for all students of anesthesia and those in other fields who would learn a little something of this specialty. We hope that it may be a useful introductory volume of interest before one proceeds to wider reading. Although we have listed only a few references as guides for further study, it has not been our intention to slight individuals who have made important contributions. Doubtless omissions and reasons for disagreement will be found.

Dr. Henry L. Price, Dr. Ronald Woolmer and others of our associates have contributed valuable suggestions. Miss Sally Van de Water has performed yeoman service as our secretarial assistant. We acknowledge these various contributions with gratitude.

ROBERT D. DRIPPS, M.D.
JAMES E. ECKENHOFF, M.D.
LEROY D. VANDAM, M.D.

September, 1957

CONTENTS

I. THE PREANESTHETIC PERIOD

<i>Chapter 1</i>	PREANESTHETIC ROUNDS	2
<i>Chapter 2</i>	PHYSICAL STATUS OR "RISK"	5
<i>Chapter 3</i>	THE CHOICE OF ANESTHESIA	7
<i>Chapter 4</i>	GUIDES TO PREANESTHETIC MEDICATION	9

II THE DAY OF ANESTHESIA

<i>Chapter 5</i>	ESSENTIAL IMMEDIATE PREOPERATIVE MEASURES	18
<i>Chapter 6</i>	ELEMENTS OF AN ANESTHETIC MACHINE	21
<i>Chapter 7</i>	COMPRESSED GASES	27
<i>Chapter 8</i>	THE MEASUREMENT OF BLOOD PRESSURE	31
<i>Chapter 9</i>	FUNDAMENTALS OF INHALATION ANESTHESIA	36
<i>Chapter 10</i>	THE INHALATION ANESTHETICS	51
<i>Chapter 11</i>	' OPEN DROP ' ANESTHESIA	61

hypnosis, the technical considerations of regional anesthesia and problems of the treatment of pain. These comprise advanced study in the field. We believe that the student for whom this book is intended should not be confronted by them until later in his training.

We trust that this book will be instructive for all students of anesthesia and those in other fields who would learn a little something of this specialty. We hope that it may be a useful introductory volume of interest before one proceeds to wider reading. Although we have listed only a few references as guides for further study, it has not been our intention to slight individuals who have made important contributions. Doubtless omissions and reasons for disagreement will be found.

Dr. Henry L. Price, Dr. Ronald Woolmer and others of our associates have contributed valuable suggestions. Miss Sally Van de Water has performed yeoman service as our secretarial assistant. We acknowledge these various contributions with gratitude.

ROBERT D. DRIPPS, M.D.
JAMES E. ECKENHOFF, M.D.
LEROY D. VANDAM, M.D.

September, 1957

V. SPECIAL TOPICS

<i>Chapter 27</i>	CARDIAC RESUSCITATION	204
<i>Chapter 28</i>	PRINCIPLES OF PEDIATRIC ANESTHESIA	211
<i>Chapter 29</i>	ANESTHESIA FOR AMBULATORY PATIENTS	219
<i>Chapter 30</i>	OBSTETRIC ANESTHESIA AND INFANT RESUSCITATION	222
<i>Chapter 31</i>	THE MANAGEMENT OF NARCOTIC POISONING	231
<i>Chapter 32</i>	FUNDAMENTALS OF INHALATION THERAPY	239
<i>Chapter 33</i>	DEATH REPORTS	247
<i>Chapter 34</i>	INSTRUCTION IN ANESTHESIA	250
<i>Index</i>		255

<i>Chapter 12</i>	FUNDAMENTALS OF MUSCLE RELAXANTS	65
<i>Chapter 13</i>	INTUBATION OF THE TRACHEA	72
<i>Chapter 14</i>	INTRAVENOUS BARBITURATE ANESTHESIA	84
<i>Chapter 15</i>	SPINAL ANESTHESIA	95
<i>Chapter 16</i>	FUNDAMENTALS OF LOCAL ANESTHESIA	116

III. DURING OPERATION

<i>Chapter 17</i>	A PHILOSOPHY OF ANESTHETIC RECORDS	124
<i>Chapter 18</i>	DETERMINATION OF THE DEPTH OF GENERAL ANESTHESIA	137
<i>Chapter 19</i>	FIRE AND EXPLOSION HAZARDS	149
<i>Chapter 20</i>	ARTERIAL HYPOTENSION DURING ANESTHESIA	155
<i>Chapter 21</i>	UNUSUAL COMPLICATIONS OF ANESTHESIA	168
<i>Chapter 22</i>	SURGEON-ANESTHETIST RELATIONSHIPS	175

IV. THE POSTOPERATIVE PERIOD

<i>Chapter 23</i>	HAZARDS OF THE IMMEDIATE POSTOPERATIVE PERIOD	180
<i>Chapter 24</i>	THE RECOVERY ROOM	188
<i>Chapter 25</i>	TREATMENT OF IMMEDIATE POSTOPERATIVE PAIN AND EXCITEMENT	193
<i>Chapter 26</i>	CLEANING AND STERILIZATION OF EQUIPMENT USED FOR INHALATION ANESTHESIA	198

V SPECIAL TOPICS

<i>Chapter 27</i>	CARDIAC RESUSCITATION	204
<i>Chapter 28</i>	PRINCIPLES OF PEDIATRIC ANESTHESIA	211
<i>Chapter 29</i>	ANESTHESIA FOR AMBULATORY PATIENTS	219
<i>Chapter 30</i>	OBSTETRIC ANESTHESIA AND INFANT RESUSCITATION	222
<i>Chapter 31</i>	THE MANAGEMENT OF NARCOTIC POISONING	231
<i>Chapter 32</i>	FUNDAMENTALS OF INHALATION THERAPY	239
<i>Chapter 33</i>	DEATH REPORTS	247
<i>Chapter 34</i>	INSTRUCTION IN ANESTHESIA	250
<i>Index</i>		255

I

The Preanesthetic Period

Preanesthetic Rounds

EVERY person who is to be given an anesthetic should be seen by his anesthetist the day before operation. When this is not possible a member of the anesthetic staff should act as a substitute and transmit his findings to the person who will give the anesthetic. Preparation for anesthesia begins at the time of this preanesthetic visit. There is no substitute for talking to a patient, listening to his problems, observing his characteristics and acquainting him with the procedure planned. Indeed, if these goals are accomplished the amount of preanesthetic medication needed to allay apprehension may be reduced. The visit is a subtle educational process for both anesthetist and patient. The patient learns what an anesthetist has to offer him, and if false concepts about anesthesia exist they can be altered. Preanesthetic rounds have much to offer in the teaching of beginners.

PROCEDURES IN PREANESTHETIC ROUNDS

The following suggestions are to be considered in making rounds. One should review the patient's past and present hospital records, focusing attention particularly on prior anesthetic experiences, physical abnormalities, personal habits, and on the physiologic alterations produced by disease. A summary of the important preoperative findings, including laboratory examinations, should be written on the anesthetic chart. Ability to tolerate the adverse effects of anesthetics and operation depends largely upon the functioning of the respiration and circulation, and the action of liver, kidneys and endocrine glands. An anesthetist should be prepared to evaluate a patient's condition along these lines. He should also discern what the proposed operation may mean from the standpoint of position on the operating table, time involved and physiologic change expected. An operative permit should be signed, particularly for minors.

When important data are lacking or further examinations are indicated, the patient's surgeon should be consulted. Rarely should there be disagreement over the safe preparation of a patient for operation. In an emergency standard practice may have to be abandoned. A patient may need anesthesia and operation even though a recent meal or an exceptionally low hemoglobin level should dictate delay. Although anesthesia is crucial, it is only a small part of the total surgical experience. Surgeon and anesthetist should devote their attention to the broad aspects of recovery from the entire experience.

When the anesthetist has accomplished these preliminaries he should have a plan of action in mind and is then ready to visit the patient. The interview with the patient must be an unhurried, tactful one with care taken to avoid irritation on both sides. Let the patient converse within limits, but stick to the point. If the patient is eating or receiving special treatments it may be better to visit at another time.

The following method of procedure is suggested for conducting the interview. Introduce yourself by name, explaining who you are and that you will provide anesthesia for the operation. The patient may not have been told by the surgeon of the final decision to operate. If so, the anesthetic procedure can be described as it may take place at any future time. Learn about previous anesthetic experiences, unusual reactions to drugs, and the type and extent of physical activity the person can tolerate. Inquire of habits, particularly prior ingestion of drugs such as barbiturates, cortisone, insulin, reserpine, digitalis, chlorpromazine, and any substances which may affect the conduct and course of anesthesia. During the interview try to assess the individual's emotional state of mind.

Search for physical characteristics which will make the administration of anesthesia technically difficult. The plethoric short-necked individual may develop respiratory tract obstruction readily, the husky athletic man usually needs a great deal of anesthetic, whereas the asthenic old woman will require little. Inquire about the condition of the teeth and remind the patient to remove dentures before coming to the operating theatre.

Once these preliminaries are over, inform the patient of the plans for anesthesia. Reactions to these suggestions will vary. Some will accede readily, others fear the face mask or the venipuncture and seek oblivion for the whole procedure. Some worry about postoperative nausea, vomiting and pain. Many fear spinal anesthesia, having heard that headache or permanent paralysis may ensue. ||

In making the final decision never be unyielding. Choose the best

anesthetic for the patient in conformity with the capabilities and preferences of anesthetist and surgeon. Most patients will accept the physician's word when confidence is inspired. In selecting spinal anesthesia be certain there are no neurologic contraindications. It is within reason to inspect and palpate the spine or caudal area, and to test the effect of the operative position on the circulation and respiration.

One should see to it that the patient secures a good night's sleep. He should be told how the preoperative medication will be given, not to eat or drink beforehand, when he will be taken to the operating area, the immediate postoperative events, and the plan of observation in a recovery room. Remind patient and relatives of the delay in returning to the ward because of the recovery room stay. Avoid discussing what is in the surgeon's domain. The patient's relatives may be troublesome in their efforts to help plan for their kin's welfare. They can be gently sidetracked.

When the anesthetist leaves the patient he should complete the preoperative section of the anesthetic record, assign a physical status classification, write the appropriate preanesthetic orders, and add to the patient's chart a note summarizing the results of the preanesthetic visit.

NOTES ON THE HOSPITAL RECORD

An anesthetist is frequently asked by an internist or surgeon to evaluate a patient. His appraisal should not be transmitted verbally, but should be added to the patient's permanent hospital record, either as part of the progress notes or on a formal consultation sheet. A consultation of real value can be written if the foregoing suggestions for the preanesthetic visit are followed. It might take the following form: a brief resume of the patient's condition and physical status, a statement of the difficulties likely to be encountered during anesthesia and operation, suggestion for preanesthetic medication, choice of anesthetic agent and technique, with reasons for each, and finally, a request for further examinations or supportives which might be required during operation.

Physical Status or "Risk"

ONE USED to refer to the surgical patient as a good or poor "risk," a term involving an estimate of prognosis from the standpoint of either mortality or morbidity. A consideration of the factors which influence the outcome in an individual case suggests that the term as ordinarily used by surgeon or anesthetist is unsound and should be abandoned. To evaluate a "risk" completely would necessitate foreknowledge of such variables as the reliability of suture material to be used, adequacy of sterilization of instruments, availability of drugs, the responsibility of those in charge of postoperative nursing care, and a host of other aspects which cannot be assessed for each patient. A patient anesthetized by a medical student and operated upon by a junior surgeon has less chance than would the same patient in the hands of an experienced anesthetist and surgeon. Under these circumstances the patient's condition has not changed, but the likelihood of survival has been materially increased. The patient's prognosis is also more favorable if the personnel responsible for his care are not tense or exhausted. Again, his condition remains the same but the risk is diminished. In addition to the patient's preoperative physical condition, therefore, the success or failure of an anesthetic or operation depends upon many unrelated factors.

Not only is the term "risk" basically incorrect, it is frequently misused. Too often a patient is characterized as a "poor risk" only *after* a catastrophe has occurred. This is a conscious or unconscious effort to cover up errors in diagnosis, management, judgment or technique. Original appraisals and decisions were at fault, or sufficiently skilled personnel were not available, yet the outcome is explained on the basis of the patient's being a "poor risk." Improvement in patient care does not lie in this direction.

CLASSIFICATION OF PHYSICAL STATUS

The term "physical status" is obviously more accurate and preferable, and can be assigned a numerical rating. The classification of the American Society of Anesthesiologists is as follows

Class 1 A patient who has no organic disease or in whom the disease is localized and causes no systemic disturbance

Class 2 A patient exhibiting slight to moderate systemic disturbance which may or may not be associated with the surgical complaint and which interferes only moderately with the patient's normal activities and general physiologic equilibrium

Class 3 A patient exhibiting severe systemic disturbance which may or may not be associated with the surgical complaint and which seriously interferes with the patient's normal activities

Class 4 A patient exhibiting extreme systemic disturbance which may or may not be associated with the surgical complaint, which interferes seriously with the patient's normal activities, and which has already become a threat to life

Class 5 A patient who is operated upon in an emergency who would otherwise be in Class 1 or 2

Class 6 A patient who is operated upon in an emergency who would otherwise be in Class 3 or 4

Class 7 The rare person who is moribund before operation, whose preoperative condition is such that he is expected to die within 24 hours even though not subjected to the additional strain of operation

At the University of Pennsylvania we see no reason to reduce four categories of physical status to two, merely because the patient has appeared as an emergency. We therefore use five classes, i.e., Classes 1-4 and Class 7 above, and add the letter E in emergency situations. Thus a patient ordinarily in Class 2 would become 2-E in an emergency.

Assignment of a numerical rating to the patient, although admittedly often difficult, pays dividends. It forces an over-all appraisal of the patient's physical condition, and focuses attention on such factors as the skill of the anesthetist or the likelihood of problems arising during anesthesia. A higher classification may alert an anesthetist to monitor a patient's progress with an electrocardiograph or to provide other safeguards. In reviewing clinical records the relationship of the anesthetic course to the physical status of the patient is always of interest. It permits easier comparison of one anesthetist's cases with those of another. The allegation that cardiac arrest occurs just as frequently in healthy surgical patients as in ill individuals can be proved only in this manner. The safety of a particular drug or technique is more readily determined with the aid of such ratings. They form an integral part of proper anesthetic management.

The Choice of Anesthesia

AN ANESTHETIST skilled in a variety of anesthetic techniques can so apply these that a particular problem may usually be solved in one of several ways. Thus, gastric resections have been and can be satisfactorily performed with any of the following: continuous spinal anesthesia, single dose spinal anesthesia supplemented with thiopental and nitrous oxide, cyclopropane, closed system ether, open drop ether; anesthesia with or without endotracheal tubes, field block with or without splanchnic block. In short, it is difficult to state categorically that there is an indication for a particular type of anesthetic for most operations or most complicating diseases. Skillfully managed, any one of a number of agents and techniques can usually be applied, the final selection often being a matter of individual preference.

The once popular examination question is unjust which presented a patient facing an operation and which required the student to select from a prescribed list the one and only anesthetic procedure that fitted the condition outlined. We are now more concerned with *why* a particular method is selected. The choice of method should be defined and fortified by *reasons* for so doing. If the latter be valid, who are we to argue the cause of spinal against general anesthesia or of intravenous versus rectally administered anesthesia?

This point of view concerns itself with the realization that the skill of the anesthetist, his knowledge of the surgeon's methods, the accepted procedures in the community, the specific request and former anesthetic experiences of the patient, must all be considered in the final choice.

This does not mean that certain indications and contraindications have not been accepted by most anesthetists. Among the latter might be listed the administration of spinal anesthesia to patients with pre-

existing neurologic disease, or of cyclopropane, thiopental or *d*-tubocurarine to patients with allergenic bronchial asthma because of the tendency toward bronchiolar constriction

It is to be expected that as knowledge of drugs used in anesthesia increases, and as one learns more about the pathologic physiology of disease, it will be possible to select the preferred anesthetic management more intelligently. The need for greater understanding of the effects of anesthetics on man has stimulated the research efforts of many workers. When the results of these programs are correlated with data obtained from the study of disease, a real advance in anesthesia will have taken place.

We do not believe that an internist called as a consultant to evaluate the physical status of a patient scheduled for operation can logically suggest the type or method of anesthesia to be used. Few internists have accompanied their patients to the operating room, have watched the problems of induction or maintenance of anesthesia, realize the difficulties posed by the bodily habitus of the patient, or appreciate the requirements of individual surgeons. Until they have practical, first-hand experience with the conduct of anesthesia it is unwise for them to insist upon a particular course, especially when dealing with anesthesiologists of modest background and experience.

Our concept of the teamwork between internist and anesthesiologist can be illustrated by what we expect of a cardiologist when asked to see a patient with heart disease. This consultant can best provide the pathologic diagnosis and assess the degree of functional impairment. He can decide whether the patient is in the best possible condition or whether further bed rest, use of diuretics, digitalis, and the like, will improve the chances for a successful convalescence. He can point out complications which may arise and how to treat them, during and immediately after operation. In short, each specialist is expected to contribute to the patient's well-being with his special fund of knowledge.

Guides to Preanesthetic Medication

DURING preanesthetic rounds the anesthetist has observed the mental and physical status of his patient and, after due consideration for the patient, the surgeon's requirements, and his own preferences, decides upon the conduct of anesthesia. Preanesthetic medication can be ordered rationally only after these observations and decisions have been made. The selection of preanesthetic drugs must be the prerogative of the anesthetist, since the administration of the anesthetic actually begins when the preparatory drugs are given in the patient's room. The proper choice of medication can pave the way to a smooth anesthetic and postoperative course. An improper choice can be the cause of an unsatisfactory anesthetic experience for all concerned.

The type and quantity of preanesthetic drugs ordered depend upon the anesthetist's goal. We believe that the patient should be an awake, alert individual. It is safer this way. Yet we make an effort to reduce apprehension. Confidence in the anesthetist helps to achieve the desired state. If confidence has been inspired during the preanesthetic visit, apprehension will usually be minimal, and the need for preanesthetic sedatives reduced. If, however, the operation poses an actual or imagined threat to the patient's well-being, confidence alone cannot be expected to allay apprehension. Sedatives then must be used. If pain is a prominent feature, analgesics are indicated.

Occasionally a patient is encountered who asks to be made unconscious before he leaves his room. The anesthetist must remember that if drugs are used to produce unconsciousness, an additional hazard is added, not only from toxic effects of the drug but from the side

effects of unconsciousness, such as respiratory obstruction. A word to the patient that the anesthetist is reluctant to use a practice that he believes is unsafe will often settle the issue.

Customarily preanesthetic drugs are used to allay apprehension, to diminish the amount of anesthetic required, to minimize autonomic reflex activity, and to reduce secretions within the respiratory tract. As will be pointed out in the succeeding paragraphs, the drugs used for these purposes have actions other than those desired. With some, the undesirable side effects are so common that the anesthetist must continually assess the price demanded by their use. In addition, he must be aware of the effect of age on drug action. A practice that may be reasonable in the young adult may be hazardous in the elderly person. Finally, he must realize that muscular and active patients require larger doses than the frail, obese or inactive.

THE PREANESTHETIC DRUGS

Drugs used for preanesthetic medication can be classified as sedatives, narcotics and belladonna derivatives.

Sedatives

Barbituric acid derivatives such as pentobarbital (Nembutal) or secobarbital (Seconal) are used immediately prior to anesthesia. When a drug with a longer action is desired, Amytal or phenobarbital may be ordered. It is well to inquire if the patient has been taking sedatives. If he is accustomed to them, the drug usually taken should be prescribed, remembering that there may be tolerance.

Barbiturates have an advantage over the narcotics in that the principal action is one of cerebral cortical depression. Data are available to indicate that for sedation, barbiturates are superior to narcotics. We prefer barbiturates to narcotics for preanesthetic sedation because in the usual doses they rarely depress the respiration or circulation (Fig. 1). Allergic responses such as urticaria and wheezing respiration are not as common as with the narcotics, while nausea and vomiting seldom follow their administration. When barbiturates are given to elderly patients, transient psychoses, agitation and excitement are common. Chloral hydrate in 0.5 to 1.0 gm. doses by mouth, and non-barbiturate sedatives such as ethnamate (Valmid) in the same doses are preferable in the aged.

If pentobarbital or secobarbital is given by mouth in 100 mg. doses, maximal sedation is reached in one to one and a half hours and will have diminished considerably in three to four hours. The action of Amytal is longer. When the short-acting drugs are injected intra-

muscularly, an effect is noted more rapidly (30 minutes) and disappears earlier (two to three hours) This is the route we customarily use With rapid intravenous injection transient hypotension and respiratory depression often follow The dosage of barbiturates for infants and children is given in Chapter 28

The several barbiturates are metabolized and excreted differently The shorter acting derivatives are conjugated principally in the liver In the presence of severe hepatic disease, narcosis from these barbitu-

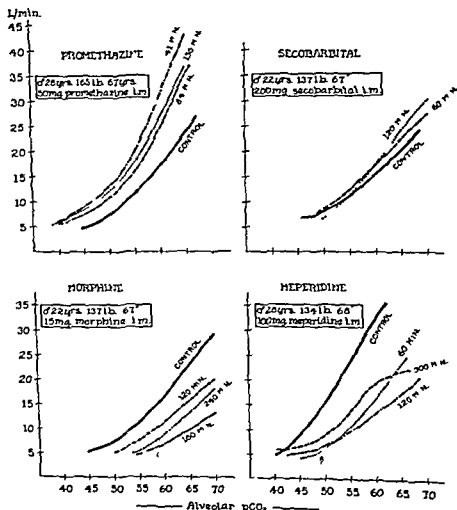


Fig 1 Comparative effects of various drugs used for preanesthetic medication upon the respiratory response to carbon dioxide in man Alveolar carbon dioxide tensions in mm. Hg are indicated on the abscissa and pulmonary ventilation in liters/minute on the ordinate The respiratory depression caused by morphine and meperidine is marked and prolonged Secobarbital was essentially without effect. The irregularities in breathing which follow promethazine (see Fig 4) caused the apparent respiratory stimulation associated with this drug (Eckenhoff Helrich and Rolph *Anesthesiology* vol. 18, 1957)

effects of unconsciousness, such as respiratory obstruction. A word to the patient that the anesthetist is reluctant to use a practice that he believes is unsafe will often settle the issue.

Customarily preanesthetic drugs are used to allay apprehension, to diminish the amount of anesthetic required, to minimize autonomic reflex activity, and to reduce secretions within the respiratory tract. As will be pointed out in the succeeding paragraphs, the drugs used for these purposes have actions other than those desired. With some, the undesirable side effects are so common that the anesthetist must continually assess the price demanded by their use. In addition, he must be aware of the effect of age on drug action. A practice that may be reasonable in the young adult may be hazardous in the elderly person. Finally, he must realize that muscular and active patients require larger doses than the frail, obese or inactive.

THE PREANESTHETIC DRUGS

Drugs used for preanesthetic medication can be classified as sedatives, narcotics and belladonna derivatives.

Sedatives

Barbituric acid derivatives such as pentobarbital (Nembutal) or secobarbital (Seconal) are used immediately prior to anesthesia. When a drug with a longer action is desired, Amytal or phenobarbital may be ordered. It is well to inquire if the patient has been taking sedatives. If he is accustomed to them, the drug usually taken should be prescribed, remembering that there may be tolerance.

Barbiturates have an advantage over the narcotics in that the principal action is one of cerebral cortical depression. Data are available to indicate that for sedation, barbiturates are superior to narcotics. We prefer barbiturates to narcotics for preanesthetic sedation because in the usual doses they rarely depress the respiration or circulation (Fig. 1). Allergic responses such as urticaria and wheezing respiration are not as common as with the narcotics, while nausea and vomiting seldom follow their administration. When barbiturates are given to elderly patients, transient psychoses, agitation and excitement are common. Chloral hydrate in 0.5 to 1.0 gm. doses by mouth, and non-barbiturate sedatives such as ethinamate (Valmid) in the same doses are preferable in the aged.

If pentobarbital or secobarbital is given by mouth in 100 mg. doses, maximal sedation is reached in one to one and a half hours and will have diminished considerably in three to four hours. The action of Amytal is longer. When the short-acting drugs are injected intra-

induction Under these conditions injection should be made slowly and it may be safer to reduce the dosage Acute respiratory depression, hypotension and vomiting may follow

The dose of morphine varies from 5 mg in older people to 15 mg in the healthy muscular male The average dose is 10 mg The corresponding doses of meperidine are 25 mg to 100 mg, and of alphaprodine 20 to 40 mg When a narcotic is combined with a barbiturate, the dose of the narcotic should be reduced Patients over the age of 65 years, and those in poor physical condition or with circulatory instability, should not be given preanesthetic narcotics because of the higher incidence of severe side effects A schedule for the preanesthetic use of narcotics in children is provided in Chapter 28

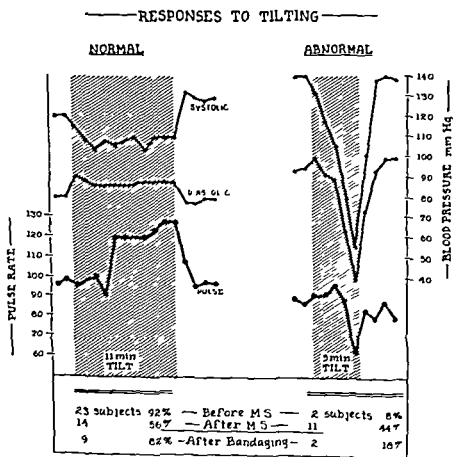


Fig 2 Postural hypotension after the intramuscular injection of morphine (M S) in man Passive tilt to the head up position revealed the circulatory depressant effect of the narcotic

rates may be exaggerated and prolonged. Phenobarbital is excreted mostly by the kidney, making this the barbiturate of choice in patients with liver disease. The function of the kidney must be ascertained before phenobarbital is ordered.

If barbiturates rather than narcotics are given preoperatively, the anesthetist may anticipate that the patient will awaken somewhat more quickly from general anesthesia, that he may perceive pain sooner, and that he may demonstrate restlessness more often as he regains consciousness. The treatment of these problems is discussed in Chapter 25. We believe the advantages of the barbiturates to outweigh the disadvantages.

Narcotics

This group comprises the alkaloids of opium and the synthetic opiate-like agents. Morphine is the opiate commonly used for pre-anesthetic medication, and in the latter category meperidine (Demerol) and alphaprodine (Nisentil) are often used. Narcotics are effective analgesics, yet pain is not a common complaint prior to operation. It has been customary to prescribe narcotics for sedation, to allay apprehension, and to diminish the amount of general anesthetic required. Data obtained in our clinic indicate that barbiturates produce better sedation and reduce apprehension more effectively.

Narcotics have many undesirable side actions. Respiratory depression is reflected in a decrease in respiratory rate, in minute volume and in the respiratory response to the stimulus of carbon dioxide (Fig. 1). The degree of depression is not detectable when respiratory rate alone is counted. A reduction in reflex control of the circulation is evidenced by an increased incidence of fainting following a 60 degree head up tilt position (Fig. 2). Vertigo, nausea and vomiting commonly appear with the assumption of the upright position. Peristalsis and intestinal tone are decreased and constipation results, sphincteric tone is increased and smooth muscle is constricted in other areas (ureter, bronchiole and common bile duct), and release of histamine is not unusual (bronchoconstriction, pruritus, and possibly hypotension). The synthetic analgesics share these side effects. It is because of these undesirable reactions that we turn to other drugs as often as possible.

Subcutaneous or intramuscular injections are preferred for pre-anesthetic medication. In the former, the optimum effect is reached in 45 to 70 minutes although respiratory depression may persist for several hours. Intramuscular injections provide a quicker onset and shorter duration. Medication may also be given intravenously shortly before

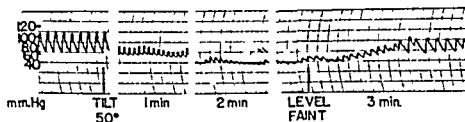


Fig 3 Continuous intra arterial recording of blood pressure showing severe postural hypotension after 50 degree head up tilt. The subject had received 50 mg chlorpromazine intramuscularly 70 minutes prior to the tilt

effect against shock of certain etiology. Evidence in support of these arguments is not convincing. These drugs predispose to postural hypotension (Fig 3). When they are given with a barbiturate or with a belladonna drug, marked restlessness may appear. One of the manifestations of this is an irregular respiratory pattern (Fig 4). Although promethazine appears to be safer than chlorpromazine, it is doubtful if the advantages of either drug outweigh the potential hazards. We do not advocate their routine use.

APPRAISAL

There is no rule in the practice of anesthesia which states that every patient should receive the same preanesthetic medication or indeed receive any at all. The anesthetist must have a reason for the choice of each drug and preanesthetic medication must be fitted to the anes-

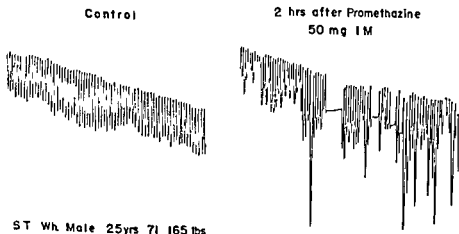


Fig 4 Respiratory spirometric tracings showing characteristic respiratory irregularities noted after the intramuscular injection of promethazine (Phenergan). The subject received 50 mg promethazine intramuscularly two hours before the tracing was taken. (Eckenhoff, Heinrich and Rolph, *Anesthesiology*, vol 18, 1957)

The question should be raised as to evidence of sensitivity to these drugs. Many patients claim that a particular substance should not be given because of alleged sensitivity. Further questioning may reveal the symptoms to have been nausea, vomiting or vertigo. Unless protracted, these are side reactions and do not represent true sensitivity. However, excitement, urticaria or production of wheezing respiration suggests that narcotics be omitted. If this is not feasible, a narcotic of totally different chemical configuration should be given.

Belladonna Drugs

These agents are given to reduce secretions within the respiratory tract and to minimize certain parasympathetic circulatory reflexes. We believe their action to be enhanced by the concomitant use of narcotics. There is no need for these drugs in patients who are to have local anesthesia unless supplementation is planned. The belladonna agents traditionally used for this purpose are atropine and scopolamine, although in recent years synthetic drugs such as methantheline (Banthine) and Antrenyl have achieved some recognition.

Scopolamine is a superior drying agent, but its use may be followed by cerebral depression and occasionally by agitation. Atropine is the choice for the cardiac and bronchial effects. Excessive dosage of either drug may lead to tachycardia. Caution must be exercised in patients with cardiac disease in whom an abrupt rise in pulse rate might predispose to pulmonary edema. Elderly patients are more likely to be excited by scopolamine.

Atropine and scopolamine are used in identical dosage. The usual dose for adults is 0.4 mg hypodermically (varying from 0.2 mg to 0.6 mg) at the time the preanesthetic narcotic or sedative is administered. The magnitude of the dose is dependent upon age, muscularity and activity. Negroes should be given a slightly higher dose because of the greater tendency to form secretions within the respiratory tract. In hot, humid weather the dose should be reduced because of the inhibitory effect on sweating and the tendency for body temperature to rise.

Other Types of Drugs

In recent years, chlorpromazine (Thorazine) and promethazine (Phenergan) have gained some popularity for preanesthetic medication. Their use is predicated on several actions: they are sedatives *per se*, they potentiate the effect of general anesthetics, they minimize autonomic reflexes, they act as antihistaminics and are said to have a protective

II

The Day of Anesthesia

thetic management If a proper relationship with the patient is established, the need for preanesthetic medication may be materially reduced Preoperative discussions and the establishment of confidence are better *and safer* than reliance on drugs to allay apprehension We prefer barbiturates to opiates primarily because of a lessened incidence of side reactions

REFERENCES

- Cohen E N ,and Beecher, H K. Narcotics in preanesthetic medication J A M A ,
147 1664, 1951
Drew J H Dripps R D and Comroe, J H Clinical studies on morphine II
Anesthesiology, 7 44 1946
Dripps R. D The pharmacological basis for preoperative medication Surg Clin
North America 24 1377, 1944
Eckenhoff J E Helrich M Hege M J D and Jones R E Respiratory
hazards of opiates and other narcotic analgesics Surg, Gynecol & Obst ,
101 701, 1955

Ask the patient when last he has eaten or drunk, whether false teeth have been removed and if he has had preanesthetic medication. Consult the nurses' record for amounts and time of administration of medications. These figures and the effect of medication should be recorded on the anesthetic chart. The degree of sedation, the circulatory and respiratory effects and any side effects should be noted.

Apply a stethoscope and blood pressure cuff to the arm remembering that both must stay in place throughout anesthesia and operation. Record initial blood pressure, pulse and respiratory rate. If there are marked changes from the expected values, an explanation for the changes must be sought. When in doubt consult the supervising anesthetist or the patient's surgeon.

An intravenous infusion should be started before anesthesia if it is needed for induction, for greater safety, or because of the patient's poor physical status. Otherwise a venipuncture is less painful for the patient after consciousness has been lost and the early vasoconstriction replaced by vasodilation. Present-day anesthetic practice sometimes requires several infusions: one for giving anesthetics, a second for fluid and blood replacement and rarely a third for special drugs. For some operations, particularly vascular procedures and radical cancer surgery, an important part of the anesthetic management lies in the anticipation, measurement and replacement of excessive blood losses. Large needles should be chosen for blood replacement. The need for venous cannulas or catheters should be anticipated in those with poor veins,

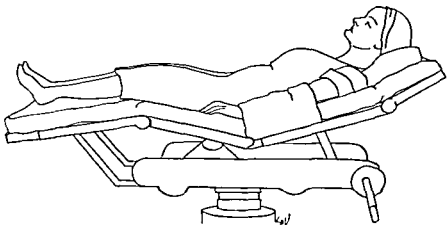


Fig 5 Flexion of the operating table for greater patient comfort. This position may be used for the cardiac patient with orthopnea in which case the back of the table is raised to a higher level.

Essential Immediate Preoperative Measures

A SAFE anesthetic can be given only if thoughtful preparations are made. The anesthetist's mental preparation begins at the time of the preoperative visit when he envisions the difficulties he may encounter and orders the preanesthetic medication.

In many instances preparing to give an anesthetic takes more time than its actual administration. For this reason we believe that the patient should be sent to the operating room about an hour before the scheduled time of operation. Having gathered equipment and anesthetic agents the anesthetist should attend to the following suggestions which apply to any anesthetic, whether local or general, and particularly for an emergency procedure.

Identify the patient and assist in his transfer to the operating table. Care in movement is most important for the very ill, the patient in pain, and for those whose circulation is precarious. Intravenous infusions, drainage catheters and traction apparatus must be guarded. The patient should be covered with a sheet or cotton blanket for warmth and for modesty's sake. A lifting sheet should be beneath the back and a broad restraining strap above the knees. Slight flexion of the operating table will often increase the patient's comfort before and during anesthesia (Fig. 5). If complete explosion precautions are practiced the patient should be grounded to the table. Patients should wear operating room caps to protect the hair, to promote sterile conditions and to keep the hair away from the face and neck. Once on an operating table a patient should not be left alone unless it is certain that he cannot fall or arise from the table.

Ask the patient when last he has eaten or drunk, whether false teeth have been removed and if he has had preanesthetic medication. Consult the nurses' record for amounts and time of administration of medications. These figures and the effect of medication should be recorded on the anesthetic chart. The degree of sedation, the circulatory and respiratory effects and any side effects should be noted.

Apply a stethoscope and blood pressure cuff to the arm remembering that both must stay in place throughout anesthesia and operation. Record initial blood pressure, pulse and respiratory rate. If there are marked changes from the expected values, an explanation for the changes must be sought. When in doubt consult the supervising anesthetist or the patient's surgeon.

An intravenous infusion should be started before anesthesia if it is needed for induction, for greater safety, or because of the patient's poor physical status. Otherwise a venipuncture is less painful for the patient after consciousness has been lost and the early vasoconstriction replaced by vasodilation. Present-day anesthetic practice sometimes requires several infusions: one for giving anesthetics, a second for fluid and blood replacement and rarely a third for special drugs. For some operations, particularly vascular procedures and radical cancer surgery, an important part of the anesthetic management lies in the anticipation, measurement and replacement of excessive blood losses. Large needles should be chosen for blood replacement. The need for venous cannulas or catheters should be anticipated in those with poor veins,

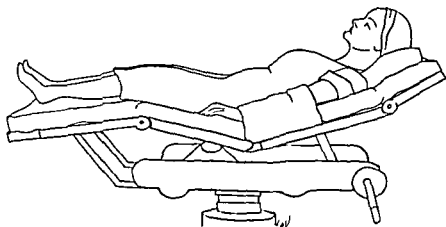


Fig. 5 Flexion of the operating table for greater patient comfort. This position may be used for the cardiac patient with orthopnea in which case the back of the table is raised to a higher level.

or with exceedingly poor physical status and for some pediatric operations

The anesthetic machine must be shown to function satisfactorily before the start of anesthesia. Gas cylinders must contain gas, flow meters must function properly, soda lime must be fresh and resistance to breathing minimal. The only way to ascertain these is to test each separately and for the anesthetist himself to breathe through the system. One should test for leaks by compressing the reservoir bag with all outlets closed.

Lastly, an appropriate face mask must be chosen for the patient. A satisfactorily fitting mask is one of the most important technical matters in inhalational anesthesia. A poor fit delays the induction of anesthesia and defeats positive pressure breathing when it is needed for resuscitation. In applying a mask avoid excessive pressure to the face and compression of the eyes. The best fit is attained by selection of a good mask and testing it on the patient's face prior to induction of anesthesia.

Artificial airways, endotracheal tubes, laryngoscope, suction catheter, bite block and gauze with lubricant should be arranged neatly on the anesthetist's work table within easy reach if necessary. Each piece of apparatus should have been demonstrated to be clean and working properly. A source of suction should be on hand and functioning before anesthesia is started.

A second person standing by can be of tremendous help during the induction of anesthesia. He can assist with restraints if there is excitement and aid in positioning the patient when special positions are required. This person can palpate the pulse at crucial times, prepare injections, start intravenous infusions and seek further help if it is needed.

The value of the foregoing routine for the operating room preparation of the patient will be appreciated only after the beginning anesthetist has encountered difficulties during induction. The extra time and preparation lead to greater peace of mind and consequently more poise in giving an anesthetic. If the anesthetist is tardy, he takes unnecessary chances and does his poorest work under the scrutiny of an impatient audience. Calmness, unhurried activity, neatness, cleanliness and punctuality will establish a reputation for a safe technique.

Elements of an Anesthetic Machine

EVERY anesthetist should be conversant with the mechanism and physical principles of an anesthetic machine. There are various makes of machines, but all have the same basic elements and design. These can be seen in the accompanying diagram (Fig. 6). The essential parts of an anesthetic machine therefore are

- 1 A source of anesthetic gases or vapors
- 2 Flow meters to measure gas flow
- 3 A reservoir bag for rebreathing
- 4 Vaporization bottles for volatile liquid anesthetics
- 5 Canisters containing carbon dioxide absorbents
- 6 Conducting tubes and face mask
- 7 Expiratory or "pop-off" valves

Supply of Gases and Vapors

Anesthetic gases are provided under increased pressure in cylinders (Fig. 6, 1) of various capacities. The cylinders, their contents and markings are standardized in the United States by the Interstate Commerce Commission. The reader should familiarize himself with the hazards of handling gases under pressure (Chapter 7) and the physical characteristics of the commonly employed anesthetic gases and volatile agents.

A cylinder should be "cracked" (opened briefly) before it is attached to the anesthetic machine. This clears dust and dirt which may otherwise foul the valve seats in the machine. Cylinders are attached to

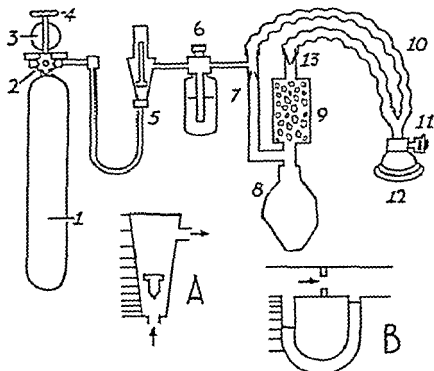


Fig 6. Elements of a typical gas machine. 1 Gas cylinder 2 Hand screw to tighten cylinder connection. 3 Pressure gauge to indicate cylinder gas content. 4 Hand wrench to open cylinder to reducing valve. 5 Flow meter 6 Vaporization bottle. 7 Inspiratory valve from reservoir bag 8 Reservoir bag 9 CO₂ absorption canister 10 Conducting tubes to face mask. 11 Expiratory pop-off valve. 12 Face mask. 13 Expiratory valve to canister A. Flow meter of the variable orifice dry type. B Flow meter of the fixed orifice wet type.

yokes, with washers of non-explosive material. The current practice of pin-indexing cylinders and yokes makes it impossible to apply a cylinder of gas to the wrong yoke. Before this precautionary measure was adopted patients were known to have been given nitrous oxide mistakenly instead of oxygen.

Each cylinder has a valve which admits the gas to the flow meters. This valve is turned on only when the flow meter valves are closed. Otherwise excessive pressures might be admitted to the delicate mechanism of the flow meters and damage them. Most anesthetic machines have pressure gauges (Fig 6 3) that indicate the pressures in the cylinder. Before anesthesia is induced the anesthetist should test the cylinders to see that a sufficient supply of gas is available. Anesthetic machines should possess double yokes so that two cylinders of oxygen, for example, are at hand. There must be provision to prevent one cylinder from filling the other. This is an important precautionary

measure since transfilling cylinders may lead to an explosion through rapid expansion and compression of gases, and possibly to the contamination of a cylinder with a gas that it should not contain. Metal or heavy rubber tubing conducts the gas from the cylinder to the flow meter (Fig 6, 5)

Flow Meters

Flow meters are present to measure the flow of gases and to allow their economical use. They differ in design from one machine to another. There are wet types and dry types. The physical principles of gas flow through the flow meter, which are based on pressure, resistance, viscosity and density, determine the scale markings through which flow is read.

A variable orifice, dry flow meter may be seen in the illustration (Fig 6, A). The gas flows from below upward through a tapered tube. As the gas flows, it raises a bobbin or float. The orifice through which the gas flows varies according to the height of the bobbin. The higher the flow, the larger the orifice. When the bobbin comes to rest the force of gravity upon it is balanced by the upward thrust of the gas.

Another type of flow meter (Fig 6, B) may be seen in the accompanying figure. As the gas flows through this fixed orifice, a pressure differential is created so that the pressure is greater on the proximal side. A good deal of kinetic energy of flow is lost because of turbulence at the orifice and the pressure distal to it is lowered. This meter has a water manometer, the limbs of which communicate with opposite sides of the orifice. Increased flow is indicated by a lowering of the water level on the proximal side and is read on a visual scale. The greater the flow the greater the depression of the water level. This flow meter is called a fixed orifice, wet flow meter.

Reservoir Bag

The flow having been measured, the gases must be delivered to the patient. A technique sometimes used is that of insufflating large quantities of the anesthetic through the patient's nose or mouth. Most anesthetics, however, are given in rebreathing systems. This technique is more economical and prevents the diffusion of explosive anesthetics throughout the operating theatre. Rebreathing also permits better control of the depth of anesthesia. Hence, a reservoir bag (Fig 6, 8) is needed in a rebreathing system. This reservoir also compensates for variations in the patient's respiratory demands. For example, if there is a constant flow of gases from the flow meter, a patient may need

more or less of the total flow depending upon the phase of respiration. At peak inspiration, gas flows of 30 to 80 liters/minute may be required, yet the flow meters may be set at a much lower level for average minute volume of ventilation of 4 or 5 liters/minute. At peak inspiratory demands the patient can obtain the greater quantity of gas needed from the reservoir bag. With lower respiratory demands gases not inhaled accumulate in the reservoir. In addition, the bag allows for resuscitation and control of pulmonary ventilation. Rhythmic compression of the bag will inflate the lungs if there is a tight mask fit about the patient's face.

Vaporization Bottles

A container (Fig. 6, 6) for the vaporization of volatile liquid anesthetics may be placed on either the inspiratory or expiratory side of the apparatus. Most gas machines are provided with adjustable valves that will allow gases to by-pass or to be directed in part or in whole through the vaporizer. Wicks provide a broader surface for vaporization of the liquid. The quantity of anesthetic vaporized depends on the gas flow through the vaporizer, the vaporizing surface and the temperature of the liquid.

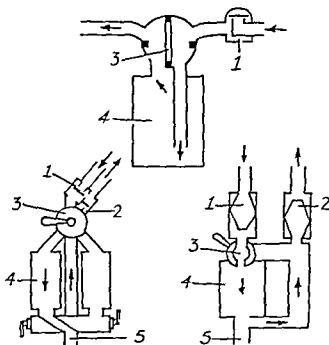


Fig. 7 Several types of circle carbon dioxide absorption canisters 1 Inspiratory valve 2. Expiratory valve 3 Canister by pass valve 4 CO₂ absorption canister 5 Connection to reservoir bag

Carbon Dioxide Absorbers

In a rebreathing system canisters of soda lime (Fig 6, 9) are necessary to absorb expired carbon dioxide. The course through the absorber may be of one or two types. Either the expired air is circulated through in one direction (Fig 7) or it passes back and forth in a to-and-fro fashion. In the former, directional valves are needed (Fig 6, 7, 13). In many anesthetic machines two canisters may be found, so that the flow of gases may be led through one or the other as the absorbent becomes exhausted. The anesthetist should constantly observe his patient for signs of carbon dioxide accumulation. These are often elusive and if there is any question about the efficiency of the absorber, it should be changed. Among other things the duration of efficient action of the absorber depends upon the patient's output of carbon dioxide, the volume of tidal air, the rate of respiration, the capacity and shape of the canister, the surface, water content and porosity of the absorbent granules, and the chemicals used for absorption.

Conducting Tubes and Face Mask

Gases must be led from the machine to patient (Fig 6, 10). Most often a face mask (Fig 6, 12) is used, but the lead tubes from the machine can be attached by adapters directly to endotracheal tubes or simply led into the mouth or nose for insufflation of large quantities of anesthetic.

Expiratory or "Pop-off" Valves

A final part of the anesthetic machine is the expiratory or "pop-off" valve (Fig 6, 11). If the flow of gases is such that the reservoir contains more anesthetic gas than the patient can breathe, provision must be made for their escape. This is the function of the 'pop-off' valve. There are several anesthetic techniques in which large gas flows are required. For example, in nitrous oxide anesthesia in order to provide a safe concentration of oxygen yet give an effective percentage of nitrous oxide, gas flows of 5 liters or more are often used. An additional reason is the need for denitrogenation of the lungs. Unless there is some means for nitrogen to escape from the rebreathing system, the partial pressure of oxygen and anesthetic gas in the alveoli will be lowered and the full anesthetic effect of nitrous oxide will not be achieved.

Having read this brief description the reader should now review and test for himself the several parts of an anesthetic machine. He will find that all anesthetic machines fit into a basic plan. The physical properties

of gases, the physics of flow meters, and factors in the vaporization of volatile anesthetics must be thoroughly understood. The anesthetic machine can be a hazard and a liability if its proper functioning is not understood. It is the knowledge and judgment of the anesthetist that make it a safe and useful device.

REFERENCE

Macintosh, R. R., and Mushin, W. W. *Physics for the Anaesthetist*. Charles C. Thomas, Springfield, Ill., 1947.

Compressed Gases

OXYGEN and certain of the gases used to produce general anesthesia are supplied in a compressed state in order that large quantities may be available in portable containers. Since these gases are supplied at greater than atmospheric pressure, several hazards are created. These are fire and explosion, and transmission of high pressures to the lungs. Persons handling compressed gases must conduct themselves in a manner designed to avoid accidents. A knowledge of the general gas laws is fundamental to an understanding of many of the problems involved in this discussion. The physical properties of the commonly used anesthetic gases may be reviewed in the accompanying table.

The manufacturer's role in safety is regulated by the Interstate Commerce Commission. The Compressed Gas Manufacturers' Association also recommends standard safety measures. Each cylinder is stamped with letters and figures, all of which have meaning (Fig. 8, C). Cylinders are designed to resist pressures at least 1.66 greater than the service pressure. A safety device in the form of an escape valve is provided against the possibility that gases may expand with high temperatures and the pressure be increased beyond safety limits. Cylinders are now pin-indexed to prevent the attachment of a cylinder to the improper yoke (Fig. 8, A).

Compression of a gas to a liquid state provides the most economical form of supply and smallest volume, but the physical characteristics of the gas limit the conditions under which this may be accomplished. Compressed oxygen as we use it is in a gaseous state because condensation into liquid requires a pressure of 50 atmospheres (735 pounds/square inch) exerted at a temperature below -118.8°C (the critical temperature above which condensation cannot occur). On the other hand, nitrous oxide is compressible to a liquid state at 50 at-

Table 1 Properties of Medical Gases

	OXYGEN	NITROUS OXIDE	CYCLOPROPANE	ETHYLENE	CARBON DIOXIDE	HELIUM
Symbol	O ₂	N ₂ O	C ₃ H ₆	C ₂ H ₄	CO ₂	He
Molecular weight	32.00	44.02	42.08	28.05	44.01	4.00
Cylinder color	Green	Blue	Orange	Red	Gray	Brown
Physical state in cylinder	Gas	Liquid	Liquid	Gas	Liquid	Gas
Specific gravity air = 1	1.105	1.531	1.481	0.974	1.529	0.138
Approximate pressure at 70° F	1650 to 2200 lbs	800 lbs	75 lbs	1250 lbs	800 lbs	1650 lbs
Critical temperature C	-118.8	35.4	124.65	9.51	31.34	-267.95
Cylinder fillings	E = 165 gals G = 1400	E = 420 gals G = 3200	D = 230 gals	E = 330 gals	E = 420 gals	E = 131 gals
Gas weight*	E = 1 lb 3.2 oz G = 15 lbs 8.5 oz	E = 6 lbs, 9 oz G = 50 lbs	D = 3 lbs 5.25 oz	E = 3 lbs, 4 oz	E = 6 lbs 9 oz	E = 2.9 oz

* Empty E Cylinder weighs 21 lbs
Empty G Cylinder weighs 110 lbs

mospheres and 28°C , a temperature which is close to prevailing temperatures. Cyclopropane and carbon dioxide behave like nitrous oxide in so far as compressibility obtains (Fig 8, B). Liquefied gases in cylinders are present partly in gaseous form because there is a constant tendency to evaporate. This increases with rises in temperature. These facts indicate why, in the case of oxygen, cylinder contents can be estimated with pressure gauges and why there is a gradual decrease in pressure as the oxygen is used. With nitrous oxide, the gas pressure remains relatively constant so long as liquid remains. Once the liquid has evaporated completely, the pressure suddenly falls. Vaporization requires heat which is extracted from the metallic cylinder and surrounding air. For this reason there is often condensation and freezing of water vapor on nitrous oxide cylinder valves when large volumes of gas have been flowing.

The following steps should be taken when cylinders are placed in service

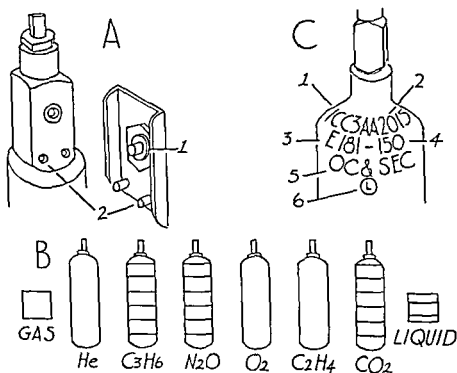


Fig 8 Features of gas cylinders A Cylinder head and yoke connection 1 Nylon washer 2 Pin index system specific for each anesthetic gas B Physical state of anesthetic in cylinder C Cylinder markings 1 Interstate Commerce Commission specifications 2 Maximum working pressure 3 Cylinder size 4 Manufacturer's serial number 5 Ownership 6 Inspector's mark

1 Paper wrappers must be removed so that identifying labels are clearly visible. Paper wrappings may catch fire or conceal the true nature of a cylinder's content.

2 Before making a connection to regulators, the cylinder valve should be opened slightly and quickly closed with the escaping jet facing away from the operator. "Cracking" a cylinder, as this maneuver is known, blows out particles of dust and other matter which might foul the mechanism of regulators and flow meters.

3 Oil, grease or inflammable liquids must not be allowed to come in contact with cylinder valves. Gases released into regulators will cool with initial expansion and generate heat within the confines of the regulator with the compression that follows. It is this secondary rise in temperature that might ignite oil or grease or combustible organic matter.

4 Cylinders should never be transfilled—that is, refilling one cylinder from another—because of the changes in temperature that may take place. Transfilling can take place when two cylinders are attached by one yoke to the same regulator. Thus the oxygen cylinders on some anesthetic machines may transfill one another if one has a higher pressure than the other and both cylinder valves are open. Valves should be fully open when in use and tightly closed when idle. Cylinders with damaged valves should be returned to the manufacturer. Empty cylinders in storage should be kept closed, to protect against intrusion of grease and dust.

5 Regulating valves must be turned off before cylinder valves are opened. The high pressures in cylinders require reduction before admission to meters that register flow rates. Most regulators are of the two-stage automatic reduction type. There is an automatic reduction of pressure in the first stage and regulation of the flow in the second. The first stage prevents damage to the delicate mechanism of the regulator. Anesthesia machines usually provide a reducing valve which must be closed as the cylinder valve is opened. Should the reducing valve be open at this time, excess pressure may distort or fracture parts of flow meters or eject water from wet flow meters. In the latter case the orifice of the flow meter may become temporarily occluded and will have to be dried before further use.

The Measurement of Blood Pressure

WE ARE still dependent on antiquated methods for determining the patient's condition during anesthesia and operation. The only criterion usually measured accurately is the respiratory rate. The heart rate can be correctly counted provided there is no discrepancy between the cardiac rate and that of the palpable pulse waves reaching the periphery. Many of the other methods by which a patient's condition can be judged are, at present, laboratory tools, and with a few exceptions have not yet been found practical or economical for daily use in every anesthetized patient. These include the electroencephalograph, the electrocardiograph, oxygen and carbon dioxide analyzers, intra-arterial and intravenous pressure recorders, and similar devices. The blood pressure apparatus usually employed (developed by Riva-Rocci over half a century ago) is by no means accurate. It is important that its proper use and its limitations be understood.

TECHNIQUE

Blood pressures should be taken from the arm that will be disturbed least during operation. This arm should be protected against contact by personnel standing around the operating table. The diaphragm of the stethoscope should be applied to the arm over the point of maximal pulsation of the brachial artery so that the sounds may be heard as clearly as possible (Fig. 9). The usual placement of the stethoscope in the antecubital space should be avoided since flexing the arm will displace it. The rubber tubings should lead to the point where the anesthetist will stand.

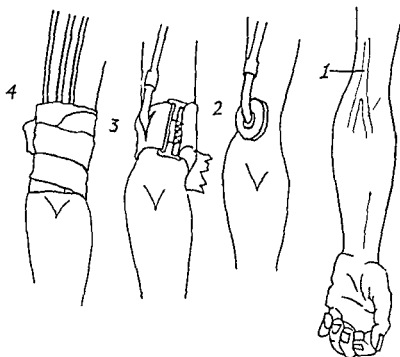


Fig 9 Placement of blood pressure cuff 1 Brachial artery 2 Stethoscope
3 Fixation of stethoscope 4 Cuff in place

The strap holding the diaphragm to the arm should be fastened as loosely as possible (consistent with keeping the diaphragm in position) in order to prevent venous congestion peripheral to the diaphragm. Venous stasis will give a falsely high diastolic pressure (+20 mm or more) and may give a false systolic pressure reading.

The cuff should be applied with the rubber bag chiefly over the inner aspect of the arm where the artery runs, and with the lower border of the bag just covering the diaphragm of the stethoscope to aid in keeping it in position. The cuff should be wound around the arm rather too loosely than too firmly so that venous flow will not be obstructed.

Before starting any anesthetic procedure, it is important to obtain a blood pressure reading which is fairly comparable to the pressures found on the ward. The effects of premedication and of psychic and other factors may alter the pressure. If the preanesthetic pressure is significantly different from that found on the ward, the cause for the difference should be sought.

The best way to obtain a blood pressure reading which is comparable to the patient's normal level is as follows: the patient should be lying supine on the level operating table so that changes due to

position will be minimal. The arm should be placed at the patient's side so that the cuff and diaphragm are approximately at the level of the heart.

An initial estimation of systolic blood pressure should be obtained simultaneously by auscultation and palpation. The pressure by palpation is usually about 5 mm below the auscultatory pressure, but it may, occasionally, be very different. The cuff pressure should be raised with a finger on the radial artery until the arterial pulsation can no longer be felt. After increasing the pressure a further 20 mm the pressure should be reduced about 2 or 3 mm a second until the first clear thumping or pounding sounds are heard. This is the *systolic pressure* by auscultation. As the pressure is lowered further, the first sounds may give way to louder sounds, sometimes accompanied by a hiss or murmur. These are then replaced by sounds similar to those first heard. As the pressure falls still lower, this clear-cut sound suddenly becomes muffled and distant. The *diastolic pressure* is the level at which this usually sudden change occurs. If this change cannot be identified, the diastolic pressure is then taken at a level where all sound disappears.

Certain factors in the use of the auscultatory method for determining blood pressure introduce errors.

- 1 If the cuff is deflated too fast the pressure readings will be grossly inaccurate. Sometimes when the pulse is not palpable an anesthetist will hastily inflate and deflate the cuff in an effort to hear one or two beats to assure himself that the heart is still beating. But if the pulse pressure is small the sounds will not be heard when the cuff is quickly deflated. When in doubt about the blood pressure, quickly ascertain if the carotid pulses are present. Never remove a blood pressure cuff to readjust it unless it be certain that there is circulation.

- 2 The auscultatory method may give confusing pressure readings if respiratory variations in arterial pressure are marked. In Figure 10 such respiratory variations are illustrated. With direct arterial recording these variations would be clearly evident. Their presence with the auscultatory method can at times be worrisome if the physiologic basis is not recognized.

- 3 If the pressure is retained in the cuff for any length of time, venous stasis will occur and the diastolic pressure will appear to be higher than it really is. The pressure in the cuff should always be completely released except for the short time required to obtain readings.

- 4 When premature beats occur, one should ignore the higher pressures of the beats that terminate a compensatory pause.

- 5 The position of the patient's arm during the operation may in-

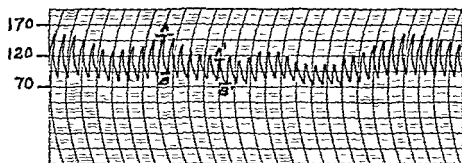


Fig 10 Variations in blood pressure caused by respiration If systolic pressure is measured at A, a higher value will be obtained than that at A, although both readings are correct Similarly, diastolic pressure would have one value at B another at B Depending upon the rate of deflation of the cuff the pulse pressure obtained by the auscultatory method could vary in this tracing from 158/75 to 112/90

introduce difficulties If the patient is lying on his side with the blood pressure arm beneath the body, there will be venous stasis, compression of the artery and the sounds will be inaudible or indistinct If the left side happens to be down and the blood pressure recorded from the arm beneath the body, heart sounds may be transmitted to the diaphragm and confuse whatever blood pressure sounds are audible If in the lateral position the patient's arm is much above the level of the heart, the diastolic pressure in the arm may be below that in the body

6 The apparatus itself may be at fault in various ways (1) aneroid barometers are easily made to be inaccurate through rough handling, (2) the level of the mercury column should be at zero level and the air vent should be free of dust or other obstruction

7 The width and length of the bag and cuff may be a source of error Correct dimensions for the average patient are given below

	INFLATABLE BAG		BAG COVER	
	Width	Length	Width	Length
Child's arm			Other dimensions in proportion	
0-1 years	2.5 cm			
1-4 years	6.0 cm			
4-8 years	9.0 cm			
Adult's arm	12 cm	23 cm	15 cm	29 cm
Adult's leg	15 cm	30 cm	17 cm	120 cm

In the obese patient it may be impossible to use the usual bag and cuff, a wider bag (18-20 cm) and longer cuff being required as in the lower leg of the average patient Conversely, for a very thin adult a smaller bag and cuff should be used If the bag is too narrow for the

size of the limb the readings will be too high, if too wide, the readings will be low. Sometimes in obese patients one is tempted to place the blood pressure cuff around the wrist or ankle. Systolic pressures obtained at these positions may be (but more often not) higher and the diastolic pressures lower than the pressures at the elbow.

Considering the many sources of error, it is remarkable how much will be revealed regarding the condition of the patient by frequent and careful observations of the blood pressure. If satisfactory control levels have been established before anesthesia and before the start of operation, subsequent blood pressure levels are a vital safeguard for the patient, the anesthetist and the surgeon.

REFERENCES

- Bordley, J. III and others. Recommendations for human blood pressure determinations by sphygmomanometers. *Circulation* 4: 503 (Oct.), 1951.
- Sapp, O. L., and Mattingly, T. W. Determination of arterial blood pressure in the lower extremity. *J. A. M. A.*, 159: 1727 (Dec. 31) 1955.
- Trout, K. W., Bertrand, C. A. and Williams, M. H. Measurement of blood pressure in obese persons. *J. A. M. A.*, 162: 970 (Nov. 3) 1956.
- Van Bergen, F. H. and others. Comparison of indirect and direct methods of measuring arterial blood pressure. *Circulation*, 10: 481 (Oct.), 1954.

Fundamentals of Inhalation Anesthesia

THEORETIC CONSIDERATIONS

GENERAL anesthetics owe their principal effects to actions on the brain that result in loss of consciousness, block of afferent nervous impulses and production of muscle relaxation. These effects occur when the blood circulating to the brain contains a sufficient amount of anesthetic. In inhalation anesthesia the anesthetic gains access to the circulation through the lungs. The success of this technique is largely dependent upon normal respiration. Hence a clear understanding of the respiratory processes at work is essential for a safe course during anesthesia.

With the exception of trichlorethylene, which is partially converted to chloral hydrate and trichloroacetic acid in the body, inhalation anesthetics are absorbed, transported and excreted without change in chemical constitution. Thus anesthetic gases and vapors are inert or non-reactive substances. They are almost entirely recoverable from the lungs save for small quantities lost by diffusion from the skin and surgical wound or through solution in the urine. They resemble other inert gases with respect to physical behavior. In other words, the general laws governing diffusion, solubility in fluids and tissues, and responses to changes in pressure and temperature apply to the gaseous anesthetics.

THE PULMONARY PHASE OF INHALATION ANESTHESIA

Inhaled Concentration

The concentration of anesthetic inspired eventually determines the level in arterial blood, therefore the amount of anesthetic which enters the brain. The concentration in arterial blood does not immediately

attain that which is being inspired, for reasons to be considered later. The anesthetist varies the mask concentration according to the needs of the operation and the signs observed in the patient. During induction a high concentration is deliberately given so that consciousness will be lost quickly and the second stage of anesthesia passed through as rapidly as possible. The modifying and limiting factors in the inspired concentration include the tightness of the mask fit and the dilution of the anesthetic with oxygen, carbon dioxide, water vapor and nitrogen. When the depth of anesthesia is to be decreased, the mask concentration is of course lessened.

Ventilation and the Residual Air of the Lungs

The air remaining in the lungs at the end of a normal expiration is called the functional residual air. The inhaled anesthetic must replace this before an effective concentration reaches the alveoli. The rate of replacement of residual air by anesthetic depends on pulmonary ventilation. With each inspiration, anesthetic is carried into the lungs. The respiratory rate and volume of the tidal air determine how quickly the residual lung air is washed out and replaced by anesthetic. If there is no rebreathing, lung washout can be accomplished in about three minutes. Lung washout and induction of anesthesia may be delayed under the following circumstances: when the minute volume of respiration is decreased by disease or by preanesthetic medication, if obstruction to respiration occurs, and in pulmonary disease such as emphysema with uneven distribution of inspired air and inadequate ventilation of certain parts of the lung.

The Pulmonary Membrane, Diffusion, and Pulmonary Blood Flow

Air in the alveoli is separated from blood in the capillaries by a thin membrane. When there is a difference in partial pressure across the two sides of this membrane, diffusion of gas occurs in the direction of lower pressure. From the start of anesthesia, anesthetics diffuse across the pulmonary membrane and are carried into the *systemic circulation* by the pulmonary circulation. The pressure gradient, the rate of blood flow, and the solubility of the anesthetic in blood influence both the rate of induction and the rate of emergence from anesthesia. Diffusion across the membrane may be slow in the presence of pulmonary edema or when the membrane is thickened by fibrosis. A sluggish circulation as in congestive heart failure may add to the effect of pulmonary edema in retarding the uptake of anesthetic.

The Pulmonary Uptake of Inhalation Anesthetic Agents

Blood leaving the pulmonary veins is normally in equilibrium with the various gases in the alveoli. For this reason, the rate at which anesthetics are removed from the lung by the blood is dependent on the rate of pulmonary blood flow, and on the solubility of the anesthetic in blood. Solubility is expressed as the partition coefficient of blood and alveolar gas concentration and has a characteristic value for every inert gas. Diethyl ether which is very soluble in blood has one of the highest values, divinyl ether an intermediate value, and nitrous oxide, cyclopropane and ethylene are among the lowest values. Actual figures for the partition coefficients of the inhalation anesthetics at 37° C are

	<u>Blood</u> <u>Gas</u>
Ethylene	0.14
Cyclopropane	0.46
Nitrous oxide	0.47
Divinyl ether	1.00
Chloroform	7.30
Ethyl ether	15.00

Because the heart and pulmonary circulation continuously carry away the inspired anesthetic, the alveolar concentration is initially prevented from attaining the inspired level. The greater the cardiac output, and the greater the partition ratio, the more slowly will alveolar concentration approach inspired concentrations and the slower will be the induction of anesthesia. Because ether has a high partition ratio, the induction time for this anesthetic is characteristically long. Induction is rapid with less soluble agents such as nitrous oxide, cyclopropane and ethylene. Induction of anesthesia with agents having a high partition ratio may be hastened by increasing the mask concentration as rapidly as possible, and by stimulating the depth of respiration with carbon dioxide. Both of these measures lead to higher alveolar concentration and a correspondingly higher blood level.

BLOOD FLOW TO ORGANS AND DISTRIBUTION OF ANESTHETICS IN TISSUES

The concentration of an anesthetic in arterial blood is important in determining the uptake of anesthetic by the brain and therefore the "depth" of narcosis. However, the blood level is constantly influenced by tissue levels in the rest of the body. While the blood flow to the brain is large per unit mass, that to liver, kidneys and heart is also great. The combined flow to these four areas comprises about 70 per cent of

the cardiac output under basal conditions. The remainder is distributed to the much larger body mass, consisting principally of muscle, fat and bone. Anesthetic is delivered to all these tissues and transferred from blood to tissue according to a partition ratio analogous to that which exists between blood and alveolar gas, although it does not have the same value. Tissues with a high blood flow rate and a low affinity for the anesthetic reach equilibrium with the blood relatively rapidly. Fat and other tissues with a low rate of blood flow may continue to absorb anesthetic for many hours. Since most anesthetics are quite soluble in fat, this represents a reservoir of high capacity which fills slowly. As time goes on and all tissues approach saturation, less anesthetic is required to maintain a given anesthetic level because less is passing into tissues from the blood. Often the anesthetic may be discontinued before the operation is completed, since the tissue stores provide such a large reservoir.

Emergence from inhalation anesthesia involves in reverse all the physiologic and physical factors heretofore discussed for induction of anesthesia. It may be looked upon as a reequilibration of the body with atmospheric air. As anesthetic is removed from the blood, the concentration in brain and other tissues is reduced, and the signs of anesthesia disappear. Awakening takes place when the blood anesthetic level falls sufficiently low. It may take a long time for all traces of anesthetic to be dissipated. Full and rapid recovery is largely dependent on normal respiration and circulation.

FACTORS APPEARING TO INFLUENCE THE AMOUNTS OF ANESTHETIC REQUIRED TO PRODUCE ANESTHESIA

It soon becomes apparent to the student that the amount of anesthetic required to produce suitable surgical conditions varies from patient to patient. In one individual, relatively small amounts of ether quickly produce first plane anesthesia, while in the next patient four or five times as much ether is required and then surgical anesthesia is obtained only after a long induction period. This is confusing to the student and requires explanation.

Under normal resting conditions, the major portion of the cardiac output is to the vital organs (brain, heart, hepatic-portal system and kidney) (Table 4). These organ systems together weigh approximately 4.6 kg, comprise only a little more than 7 per cent of the body weight, yet receive approximately 70 per cent of the cardiac output. The remaining 93 per cent of the body mass therefore is supplied by only 30 per cent of the cardiac output (Table 5).

Table 4 Regional Blood Flow and Oxygen Consumption

REGION	WEIGHT (kg)	PERCENT- AGE OF TOTAL WEIGHT	ORGAN BLOOD FLOW (cc/min)	CARDIAC OUTPUT (%)	ORGAN OXYGEN UPTAKE (cc/min)	TOTAL OXYGEN UPTAKE (%)
Brain	1.4	2.2	750	13.9	46.2	22.9
Heart	0.3	0.5	255	4.7	29.1	11.6
Hepatic portal	2.6	4.1	1500	27.7	51.0	20.4
Kidney	0.3	0.5	1260	23.3	17.6	5.0
Skeletal muscle	31.0	49.2	840	15.6	49.6	20.0
Skin	3.6	5.7	460	8.4	10.9	4.8
Residual tissue	23.8	37.8	380	6.4	50.0	15.3

Adapted from H. C. Bazett: *Medical Physiology*, C. V. Mosby Co., St. Louis, 1956, p. 221.

Table 5 Comparison of Regional Blood Flow and Oxygen Consumption to Vital Organs and Other Tissue

REGION	WEIGHT (kg)	PERCENT- AGE OF TOTAL WEIGHT	ORGAN BLOOD FLOW (cc/min)	CARDIAC OUTPUT (%)	ORGAN OXYGEN UPTAKE (cc/min)	TOTAL OXYGEN UPTAKE (%)
Vital organs (Brain, heart, hepatic portal kidneys)	4.6	7.3	3765	69.6	143.9	59.9
Other tissues	58.4	92.7	1680	30.4	110.5	40.1

The relative proportion of the cardiac output delivered to these organ systems changes under various circumstances. Thus, under conditions of muscular activity, that proportion delivered to muscle may increase many fold. During digestion, the hepatic-portal system receives a greater supply. In hot weather the cutaneous circulation is more active. Under conditions of stress, the liberation of epinephrine leads to increased muscle blood flow. In shock, hemorrhage or lowered blood volume from any cause, circulation to the periphery is sacrificed to maintain that to the vital organs.

We have discussed the fact that general anesthetics produce anesthesia by virtue of their being delivered to the brain in high concentration. Despite this, relatively large amounts of anesthetic must be

given during the induction and early maintenance phase in the normal patient because of recirculation of anesthetic and its deposition in other body tissues, particularly fat. As these depots become saturated, smaller amounts of anesthetic are required to maintain anesthesia since equilibrium or near equilibrium has been established between brain, blood and other tissues.

It follows that anything influencing peripheral blood flow, particularly that to fat and to muscle, will likewise influence the amount of anesthetic required to induce and maintain anesthesia. In addition, most general anesthetics diminish or temporarily inhibit renal blood flow. In conditions of shock or diminished blood volume, only small amounts of anesthetic may be required, since the large depots that normally have to be saturated are of no concern by virtue of the fact that circulation within them is markedly reduced or absent. Under these circumstances it is easy to administer a relative overdose of anesthetic, and produce severe cerebral or cardiac depression.

Conversely, when peripheral blood flow is unusually high as it may be in the muscularly active or in the apprehensive individual, the brain may receive a somewhat smaller proportion of the cardiac output and thus the anesthetic. Recirculation becomes more active and uptake of anesthetic by non-nervous tissue more rapid. Induction of anesthesia is thereby slowed and larger than usual quantities of anesthetic may be required until equilibrium is reached. In addition, struggling during induction increases muscle blood flow and liberation of epinephrine, and increases the amount of anesthetic required.

Patients in whom a slow induction and the need for excessive amounts of anesthetic may be anticipated include healthy muscular persons, apprehensive patients, those with thyrotoxicosis, and the young and physically active. A more rapid induction and less anesthetic may be expected in the patient in shock, and in the chronically ill, debilitated, sedentary or aged. The sicker the patient, the less anesthetic required.

Factors other than organ blood flow also appear to influence anesthetic requirements. It is possible that these factors also directly or indirectly influence blood flow but if so, the relationship has not been clarified. It is well known that chronic alcoholics are tolerant of both inhalational and intravenous anesthetics, in fact, prolonged inductions and wild excitement are so common that spinal anesthesia is usually the method of choice where applicable. There are also data to suggest that preanesthetic narcotics and sedatives will reduce the anesthetic concentration in the blood required to produce and maintain anesthesia.

This would appear to be a phenomenon of supplementation of narcosis rather than by a direct influence upon the distribution of anesthetic within the body. Narcotics administered during inhalational or intravenous anesthesia usually reduce the anesthetic requirement.

THE NEUROPHYSIOLOGIC BASIS OF ANESTHESIA

The manner in which anesthetics depress activity in the central nervous system has provoked much thought. Though it is not the intention to discuss theories of narcosis, some explanation might be offered for the characteristic order of depression seen during anesthesia. Areas of the brain seem to be depressed in the order of phylogenetic appearance, the newer areas being more sensitive. The latter are also most sensitive to oxygen deprivation. It has been assumed, though by no means proved, that metabolism differs among the several areas of the brain and that interference with enzymes concerned in oxygen utilization is more readily accomplished in the higher centers. This may not be the reason why depression with anesthetics occurs, however, since recent studies suggest that inhibition of enzymatic action and consequent decreased tissue respiration may be the result of rather than the reason for narcosis.

A neurophysiologic explanation for anesthesia may lie in the effect of anesthetics on the reticular activating substance of the brain stem. This is an area adjacent to many peripheral sensory pathways and one influenced by other regions of the brain through complex synaptic connections and associated pathways. Stimulation of this region causes arousal of a dormant animal whereas destruction leads to chronic unresponsiveness. Afferent impulses may go through this polysynaptic reticular activating system but also take a more direct pathway to the thalamus, a route that is less influenced by synaptic connections and associated regions of the central nervous system. This latter pathway has been termed the afferent lemnisco-thalamic system. Studies in animals by Magoun and French have shown that pentobarbital exerts its principal effect upon the reticular activating system (Fig. 11), tending to depress its electrical activity. In contrast, the effect upon the afferent lemnisco-thalamic system is minimal except where large concentrations of the drug were present. Ether differs from pentobarbital in that it has a greater effect upon the afferent lemnisco-thalamic system. Cyclopropane, ethylene and nitrous oxide have been shown by Davis and co-workers to have similar effects which parallel their potencies. The block of impulses from the periphery may explain in part at

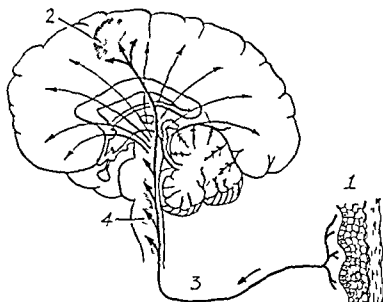


Fig 11 The reticular formation (After J D French, Scientific American May 1957) 1 Sense organ 2 Sensory area in brain 3 Sensory pathway 4 Reticular formation

least the analgesia that can be produced by small concentrations of the latter three agents

PRACTICAL APPLICATION

Preparation of the Patient for Inhalation Anesthesia

The patient about to undergo inhalation anesthesia should be prepared according to the suggestions made in Chapter 5. A strap to assist in holding the face mask should be placed under the occiput and the patient should be properly restrained. It is especially important that the induction room be quiet, that a second person stand by for assistance, and that suction be readied to cope with vomiting. It is the anesthetist's responsibility to avoid injury to superficial nerves from pressure and stretch, injury to the face and eyes from mask straps and injury from stretch of the brachial plexus. He must guard against trauma to lips and teeth. A soft pillow under the knees and one at the small of the back may prevent postoperative discomfort from strain. The same effect may be obtained through slight flexion of the operating table.

Technique

Induction As the mask is applied and the patient begins to lose consciousness he should be encouraged and lulled into a state of se-

curity by the anesthetist. It is wise not to concentrate the patient's attention upon physiologic processes such as breathing, but to explain what is happening and to suggest a tranquil induction and emergence from anesthesia. This type of hypnotic suggestion adds considerably to the ease and safety of induction.

Airway and Respiratory Problems The most troublesome problems in inhalation anesthesia naturally involve the airway and respiration. In their probable order of development they may be encountered as follows:

1 **THE MASK FIT** If a mask is fitted poorly to the patient's face the effective concentration of anesthetic given will be diluted by the inhalation of air. A good mask fit is hard to achieve in patients who are endentulous, those with prominent noses or receding jaws, and those with gastrointestinal tubes protruding from the nose or mouth. Likewise, a poorly fitted mask will make it difficult to apply positive pressure for inflation of the lungs should it be needed. These deficiencies prevent a smooth and orderly induction as well as maintenance of an even plane of anesthesia.

2 **DEPRESSED RESPIRATION** If respirations are depressed during induction of anesthesia by inhalational agents, the alveolar concentration of anesthetic may not reach that needed to produce anesthesia. Loss of consciousness or sufficiently deep anesthesia then fails to materialize. Overdose with preanesthetic medication is the most common cause for this complication. The depression of respiration may not be evident until the first few breaths of the anesthetic are taken. Intravenous barbiturates for the induction of anesthesia and too early administration of muscle relaxants, both of which hinder respiration, will also interfere with induction of anesthesia.

3 **ABNORMAL RESPIRATORY PATTERNS AND LUNG DISEASE** Rapid shallow respirations or tachypnea may not provide the alveolar ventilation needed for the uptake of anesthetics by the lungs. Diseases of the lung characterized by poor mixing of gases in the alveoli, unequal distribution among the parts of the lung or slow diffusion through the pulmonary membrane into the capillary blood will also interfere with inhalation anesthesia. Certain types of heart disease with changes in the circulation and respiration will have the same effect.

4 **RESPIRATORY OBSTRUCTION** Almost as soon as a patient loses consciousness while in the supine position, the lower jaw relaxes and recedes. The jaws may be clenched in the excitement stage. In either case the tongue may cause obstruction as it is sucked against the hard palate during inspiration, or when it falls back into the oropharynx. This is



Fig 12 Technique of lifting jaw with fingers behind the mandible to overcome respiratory soft tissue obstruction

indicated by stertorous or snoring sounds, or may be detected by listening closely over the rebreathing tubes. Respiratory obstruction is best discovered, however, by observing the rise and fall of the chest rather than the movement of the rebreathing bag. In the presence of obstruction the chest will retract as the diaphragm descends, whereas it should expand. This gives rise to a characteristic rocking motion and reveals that the descent of the diaphragm is not followed by a free inflow of air. Although movements of the rebreathing bag do not indicate the adequacy of pulmonary ventilation, failure of the bag to fluctuate indicates that the anesthetic is not being breathed. Respiratory obstruction must be corrected at once.

When obstruction is diagnosed the jaw should be lifted upward, thereby displacing the tongue. Extension of the cervical spine or turning the head to the side may help occasionally. An effective way of lifting the jaw is to place the fingers behind the vertical ramus of the mandible (Fig 12). This maneuver is one of the most difficult to teach the beginner. If applied it should be maintained for as short a time as possible since the patient may develop localized soreness and swelling.

It is possible beforehand to single out those who will develop upper respiratory tract soft tissue obstruction during anesthesia. Topical local anesthesia applied to the mucous membranes of the nose and pharynx will make the early placement of artificial airways easier. An artificial airway should be inserted only if indicated but as soon as practical. Besides endotracheal tubes, which are discussed in another chapter, there are two main types of airway, the oropharyngeal and nasopharyngeal (Fig 13). The purpose of either airway is to displace the tongue

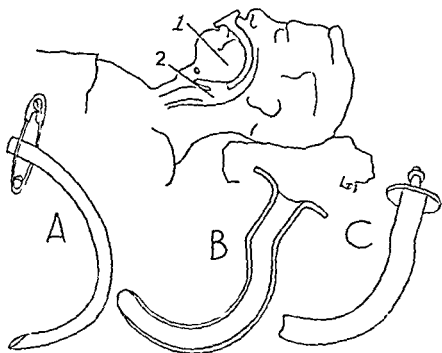


Fig 13 Pharyngeal airways Oropharyngeal airway displacing tongue (1) forward and upward from the pharynx (2) A. Nasopharyngeal airway of soft rubber tubing B Plastic oropharyngeal airway C Hard rubber oropharyngeal airway with nipple for oxygen insufflation

anteriorly. The patient breathes through and around the airway. While the oral type is more efficient, obstruction may take place before the jaw relaxes and the mouth cannot be opened to place the airway. In this situation, a soft, well lubricated nasopharyngeal tube should be passed. Force must be avoided because the nose is lined with a delicate, highly vascular mucous membrane and hemorrhage from trauma may be severe. The oral airway should be placed just as carefully, to avoid injury to lips and teeth. It can be inserted most easily if moistened with water or lubricated. Proper placement of an airway requires skill. It must be done quickly and deftly. Speed is essential during insertion so that anesthetic is not lost from the lungs while the face mask is removed. At the same time the reservoir bag should not be allowed to lose its contents, since it may then be refilled with an incorrect mixture.

5 COUGH AND LARYNGOSPASM Cough frequently follows the placement of an airway. This may be due to pharyngeal stimulation during light anesthesia or merely to irritation of the respiratory passages from a higher concentration of anesthetic made possible by a better airway.

It may be avoided by decreasing the concentration of anesthetic after the airway is inserted and then increasing it slowly. Cough can be overcome by deepening anesthesia and assisting the inspiratory phase of the cough through pressure on the reservoir bag.

Laryngospasm is a severe complication at any time but probably of most consequence during induction of anesthesia. Laryngospasm occurs most frequently in light planes of anesthesia when there is direct pharyngeal, laryngeal or peripheral painful stimulation. All degrees of laryngospasm may occur from a minor type indicated by a high-pitched crowing sound to complete, impassable closure. Sustained moderate pressure on the rebreathing bag may help in overcoming spasm. The assistant should keep his finger on the pulse to detect signs of a failing circulation indicated often by a progressive slowing and loss of amplitude of the pulse. If laryngospasm is complete and lasts for as long as a minute or so, the intravenous injection of a small dose (40 mg) of succinylcholine will relax the striated muscles of the larynx.

6 MUCOUS AND SALIVARY SECRETIONS Accumulation of secretions in the air passages can produce obstruction. Increase in secretions may be part of the initial cerebral stimulant phase of general anesthesia, or may be due to direct irritation from the anesthetic which is further increased by anoxia and retention of carbon dioxide. A major purpose of preanesthetic medication is to decrease secretions. Blocking vagal activity with atropine eliminates the profuse watery type of secretion and leaves a sparse, viscid and glairy one. If secretions are troublesome the patient's head may be turned to the side and lowered to allow escape through the side of the mouth. If a real problem, suction of the pharynx should be performed.

7 RETCHING AND VOMITING Anesthetic stimulation of the vomiting center in the medulla occurs during induction and emergence in most cases. Opiates and similar drugs used for preanesthetic medication, and subsequent movement of the patient, increase the possibility for its occurrence. The anesthetist can cope with this problem only by constant anticipation and watching for premonitory signs. The hand holding the face mask may detect the swallowing, breath-holding and retching that precede vomiting. The head should be turned to the side and the mask lifted to detect vomitus. Vomitus should be aspirated from the pharynx before the plane of anesthesia is deepened. Aspiration of vomitus into the trachea will not be a major problem if the patient has been properly prepared. Fortunately in most cases, vomiting occurs when tracheal reflexes and cough are present.

The aforementioned difficulties and complications may be encountered in rapid succession, and frequently simultaneously, during induction of anesthesia. There are added problems of prolonged excitement with strong muscular movements, breath-holding and phonation. It takes considerable experience to avoid these complications as well as to develop mastery in handling them when they appear. They will be encountered most commonly during anesthetization with the nitrous oxide-ether sequence because of the long induction period. The beginner, in using this sequence, will experience all of these problems, and will become a better anesthetist for it.

Maintenance Early in the anesthesia it is best to produce a plane of anesthesia somewhat deeper than that demanded by the proposed operation. It is easier to lighten anesthesia than to deepen it. Subsequently the concentration of anesthetic agent can be decreased. Until it has been demonstrated that anesthesia is adequate for the operation, it is usually better to err on the deep side.

A satisfactory level of anesthesia for the start of the operation having been produced, the lightest plane of anesthesia compatible with good operating conditions should be maintained. The less drug administered the better, yet too little anesthesia defeats the purpose, prolongs the operation and often leads to excessive anesthetic being given. When greater relaxation is required (as during closure of the peritoneal cavity), the need should be anticipated and anesthesia deepened at the proper time.

The anesthetist can obtain invaluable information by observing the surgical field throughout the operation. The following observations may be made: (a) The adequacy of oxygenation through the color of the blood in the wound. This may be the best means of detecting cyanosis, particularly in dark-skinned persons. (b) The amount and character of bleeding. Little bleeding suggests poor circulation. Continuous ooze may mean a clotting defect or a transfusion reaction. Excessive bleeding should alert one to the need for transfusion. (c) The degree of muscular relaxation. More or less relaxation may be needed at specific times during the surgical procedure. The caliber and tonicity of the bowel and its protrusion into the wound are indicative of the depth of anesthesia. A surgeon should not have to tell the anesthetist that greater relaxation is needed. The anesthetist can detect the need and should make the necessary adjustments. (d) Observation of the surgeon's manipulations. Packs in the abdomen, traction on viscera or sudden decompression of the abdomen as in ascites or in the delivery of a large ovarian tumor may

lead to sudden hypotension. The anesthetist should be aware constantly of what the surgeon is doing.

It is good to have patients as nearly awake as possible at the termination of the operation. If laryngeal and pharyngeal reflexes have returned, the patient is less likely to develop respiratory obstruction or to aspirate stomach contents if he should vomit. However, the desire to have a patient awake should not lead to so little anesthesia that the patient must be restrained while the last skin sutures are placed. This too can be hazardous for the patient.

Emergence During emergence those complications described under induction of anesthesia may reappear. Additional complications can occur in the immediate postoperative period (Chapter 23). When it is considered safe to move the patient, the anesthetist should accompany him to the recovery room. *The transfer to bed or litter must be gentle.* If still unconscious the patient should lie on his side to protect against airway obstruction or aspiration of vomitus. Before leaving the patient the vital signs must be determined once more and all important information with regard to treatment should be transmitted to recovery room personnel.

APPRAISAL

The theoretical and practical aspects of inhalation anesthesia have been treated in this chapter. This is a controllable technique because the lungs act as the avenue of entrance and escape for the anesthetic. The patient's respiratory efforts or the anesthetist's artificial control of respiration determines the level of anesthesia from moment to moment. Yet greater skill and knowledge are required to give an inhalational anesthetic than for intravenous or rectal drugs. Preanesthetic medication must be chosen with a view to disturbing respiration and circulation as little as possible. Respiratory obstruction from soft tissues, excessive secretions, and laryngospasm must be avoided or treated promptly if induction of anesthesia is to be rapid and safe. The phases of normal respiration must be clearly understood. Abnormalities in ventilation and diffusion must be detected for their influence on the course of anesthesia. The role of the circulation and the body tissues as they relate to the distribution of anesthetic in the brain must be clearly in mind. The physical properties of gases relating to solubility and diffusion must be understood. For all the knowledge required, we believe that inhalation is the best technique for the beginner in that it will teach him most. Once this technique is mastered all others will be simple in comparison.

The aforementioned difficulties and complications may be encountered in rapid succession, and frequently simultaneously, during induction of anesthesia. There are added problems of prolonged excitement with strong muscular movements, breath-holding and phonation. It takes considerable experience to avoid these complications as well as to develop mastery in handling them when they appear. They will be encountered *most commonly during anesthetization with the nitrous oxide-ether sequence* because of the long induction period. The beginner, in using this sequence, will experience all of these problems, and will become a better anesthetist for it.

Maintenance Early in the anesthesia it is best to produce a plane of anesthesia somewhat deeper than that demanded by the proposed operation. It is easier to lighten anesthesia than to deepen it. Subsequently the concentration of anesthetic agent can be decreased. Until it has been demonstrated that anesthesia is adequate for the operation, it is usually better to err on the deep side.

A satisfactory level of anesthesia for the start of the operation having been produced, the lightest plane of anesthesia compatible with good operating conditions should be maintained. The less drug administered the better, yet too little anesthesia defeats the purpose, prolongs the operation and often leads to excessive anesthetic being given. When greater relaxation is required (as during closure of the peritoneal cavity), the need should be anticipated and anesthesia deepened at the proper time.

The anesthetist can obtain invaluable information by observing the surgical field throughout the operation. The following observations may be made: (a) The adequacy of oxygenation through the color of the blood in the wound. This may be the best means of detecting cyanosis, particularly in dark-skinned persons. (b) The amount and character of bleeding. Little bleeding suggests poor circulation. Continuous ooze may mean a clotting defect or a transfusion reaction. Excessive bleeding should alert one to the need for transfusion. (c) The degree of muscular relaxation. More or less relaxation may be needed at specific times during the surgical procedure. The caliber and tonicity of the bowel and its protrusion into the wound are indicative of the depth of anesthesia. A surgeon should not have to tell the anesthetist that greater relaxation is needed. The anesthetist can detect the need and should make the necessary adjustments. (d) Observation of the surgeon's manipulations. Packs in the abdomen, traction on viscera or sudden decompression of the abdomen as in ascites or in the delivery of a large ovarian tumor may

The Inhalation Anesthetics

WHEN administering drugs sufficiently potent to produce unconsciousness, one must be concerned with the *controllability* of the methods involved. Ideally, one should be able to regulate the concentration of a general anesthetic in the blood stream from moment to moment. If more be needed, the concentration should be promptly increased, if overdose is evident, the concentration should be as promptly decreased.

The three methods of general anesthesia—rectal, intravenous and inhalation—vary considerably in controllability. From the standpoint of absorption the intravenous and inhalation routes remain under one's control, since in the former injection is made directly into the blood stream and in the latter, because of the enormous absorptive surface of the lungs, changes in alveolar anesthetic concentration are as a rule instantly reflected in the blood. Absorption into the blood stream from rectum and colon is, however, uncertain and unpredictable.

From the standpoint of *elimination*, the majority of drugs used for intravenous and rectal anesthesia are changed in varying degree in the body by oxidation, reduction, hydrolysis or conjugation. The safety of these substances is, therefore, related to the adequacy of specific bodily detoxification processes. Once they are in the blood stream there is no way of prompt removal unless future research develops a rapid means of hastening their metabolism. Urinary excretion is slow and uncontrollable. Inhalation anesthetics, except for trichlorethylene which is partially converted to chloral hydrate and trichloroacetic acid, are not altered in the body. They may be recovered unchanged in expired air. To prove this point, expired gases can be collected from one patient and used to anesthetize another. With inhalation anesthesia the uptake and elimination of drugs are accomplished by pul-

REFERENCES

- Davis, H S , Collins, W S , Randt, C T , and Dillon, W H Effect of anesthetic agents on evoked central nervous system responses Gaseous agents *Anesthesiology* 18 634, 1957
- French, J D Reticular Formation *Scientific American*, May, 1957
- French, J D , Verzeano, M , and Magoun, H W A neural basis of the anesthetic state *Arch. Neurol & Psychiat.* 69 519, 1953
- Haggard, H W The absorption, distribution and elimination of ethyl ether Series of five papers *J Biol Chem* 59 737-802, 1924
- Kety, S S The physiological and physical factors governing the uptake of anesthetic gases by the body *Anesthesiology*, 11 517, 1950
- Saunders, P An analysis of inhalation anesthesia by graphic means *Anesthesiology*, 5 274, 1944
- Vandam, L D Uptake of Anesthetics and Signs of Anesthesia, in *Drill, V E Pharmacology in Medicine* McGraw Hill Book Co , New York, 1954

propane anesthesia, and at the level of respiratory arrest blood pressure is higher than with ether anesthesia. This characteristic of maintenance of blood pressure makes cyclopropane the choice of many anesthetists for the anesthetic management of patients in shock. A rise in arterial pressure is not infrequent in clinical practice. This occurs more often during operation and in light planes of anesthesia. One might postulate that the increase in blood pressure is related to afferent impulses arising at the operative site. Respiratory acidosis may also contribute to the hypertension.

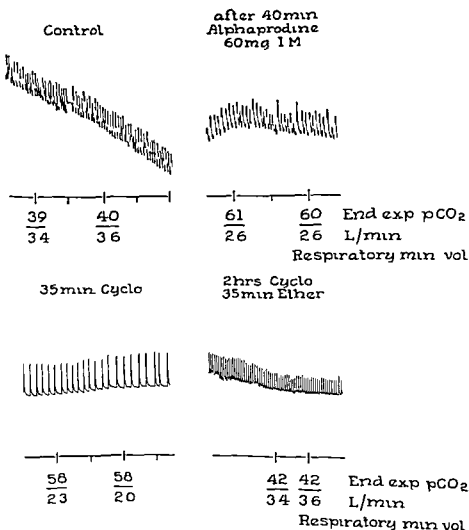


Fig 14 The respiratory depression of cyclopropane in man particularly after use of a narcotic for preanesthetic medication is indicated by the rise of end-expiratory carbon dioxide tension and the reduction of respiratory minute volume. The respiratory stimulant action of ether is shown by a reversal of these two changes.

monary ventilation. This is thus the most controllable of the methods used to produce general anesthesia.

Inhalation anesthesia is provided by volatile liquids and gases. The established drugs include the following:

GASEOUS ANESTHETICS

Cyclopropane

Nitrous oxide

Ethylene

VOLATILE LIQUID ANESTHETICS

Ethyl ether

Divinyl ether

Trichlorethylene

Ethyl chloride

Chloroform

All of these compounds are flammable except nitrous oxide, trichlorethylene and chloroform.

Certain basic principles of the pharmacologic action of the inhalation anesthetics will be presented, with emphasis on ether and cyclopropane. For a more detailed consideration the student is referred to textbooks of pharmacology.

CYCLOPROPANE

Cyclopropane or trimethylene, the simplest cyclic hydrocarbon, is the most potent of the gases used for inhalation anesthesia. It is administered in inspired concentrations ranging from 5 to 25 per cent, and has a wide margin of safety between anesthetic and toxic concentrations if respiratory adequacy is maintained. As a rule induction of anesthesia is rapid and smooth.

Cyclopropane differs from most of the volatile liquid anesthetics in that it produces little respiratory stimulation. The minute volume of ventilation is reduced as the depth of cyclopropane narcosis increases. This progressive respiratory depression is useful as an index of the level of anesthesia. If narcotics are used for preanesthetic medication, cyclopropane causes marked respiratory depression or apnea, even in light planes of anesthesia. Barbiturates are preferable for preanesthetic medication, therefore, if sedation is sought prior to induction. Because of the ease of production of respiratory depression, respiratory acidosis is a common accompaniment of cyclopropane anesthesia. It is important, therefore, to ensure adequate alveolar ventilation by manual compression of the breathing bag as tidal volume begins to diminish. The addition of small amounts of ether—a respiratory stimulant—also tends to increase respiratory activity (Fig. 14).

Arterial blood pressure tends to be well maintained during cyclo-

concept arose that cyclopropane—and other hydrocarbons such as carbon tetrachloride or benzene—"sensitized" the myocardium or made it more "irritable." Evidence for the ill effects of epinephrine in man is scant because most investigators have feared to study the problem under controlled conditions. Epinephrine does not appear to be liberated by cyclopropane in man, but it has been shown that respiratory acidosis is followed by a rise in peripheral blood epinephrine and norepinephrine in the human. The appearance of cardiac irregularities in the presence of decreased alveolar ventilation may be explained on this basis.

The effects of cyclopropane on renal and hepatic function are minimal. Data obtained in animals suggest that the drug possesses parasympathomimetic properties. For this reason cyclopropane is not administered by some to patients with allergic bronchial asthma. We regard this as only a relative contraindication.

Cyclopropane remains a controversial anesthetic. We like its controllability, potency and patient acceptance. We rely on it frequently for the anesthetic management of critically ill patients. There is increasing evidence in man of the safety of the drug and we believe that it merits more widespread use.

NITROUS OXIDE

Nitrous oxide is the only inorganic gas used as an anesthetic. Of the general anesthetics, this substance causes the least disturbance to bodily function if it is given with a concentration of oxygen equal to or greater than that in air (21 per cent). Brain damage reported after the inhalation of this substance is caused by hypoxia. The onset of and emergence from anesthesia are rapid and usually uneventful.

Nitrous oxide is a weak anesthetic, but its lack of potency can be overcome in several ways. Preanesthetic medication with narcotics can be provided in doses larger than those used with the more powerful inhalation anesthetics. Additional amounts of these substances can be injected intravenously during anesthesia. Basal narcosis with Avertin or thiopental given rectally may be used. Intravenous barbiturates are common adjuncts for supplementation of nitrous oxide. The failure of barbiturates to provide analgesia is balanced by the ability of nitrous oxide to block afferent impulses from the operative site (p. 42). If relaxation is required, one of the muscle relaxants must be added to any of the above techniques. All of these methods permit use of the electrocautery. For the patient in profound shock, nitrous oxide in concentrations as low as 50 per cent will often provide adequate anesthesia.

Systemic venous pressure rises progressively as the depth of cyclopropane anesthesia increases. This venous hypertension may be related to bradycardia, myocardial depression, or the rise in arterial pressure noted above, the last requiring increased filling pressure of the heart in accordance with Starling's Law of the Heart.

In common with most inhalation anesthetics studied in man, cyclopropane causes a considerable redistribution of blood flow, at least early during its administration. Total cardiac output is not markedly reduced in the planes of anesthesia used for surgical operations. Skin and muscle blood flow increases while renal flow is reduced. In the few studies recorded to date, hepatic flow also was reduced. The tendency toward dilation of skin and muscle vessels and the elevation of venous pressure may explain the alleged tendency of cyclopropane to increase capillary oozing.

A decline in blood pressure below preanesthetic values may be seen at the conclusion of cyclopropane anesthesia. The causes of this response have not been documented completely, but a greater degree of hypotension has been shown to follow carbon dioxide retention during anesthesia. This does not appear to be the only factor involved. Other possibilities are listed in Chapter 23.

Irregularities of cardiac rhythm occurring during cyclopropane anesthesia are a cause of concern to many. Observations in our laboratory indicate that cardiac rate tends to remain in the range of 60 to 70 per minute, and few abnormalities of rhythm are noted in healthy patients receiving cyclopropane if the depth of anesthesia does not exceed second plane and if carbon dioxide is not permitted to accumulate. Disorders of rate and rhythm begin to appear as the depth of anesthesia increases or respiratory acidosis develops. These cardiac changes usually take the form of ventricular extrasystoles, or less commonly ventricular tachycardia with rates up to 180 per minute. The latter may be a rapid, regular rhythm, or if there are multiple foci of irritability in the ventricle, the rate may be rapid and the rhythm quite irregular. This irregular rhythm is difficult to distinguish from auricular fibrillation by palpation of the peripheral pulse. It is claimed, as the result of studies in animals, that ventricular tachycardia is the precursor of ventricular fibrillation. The incidence of ventricular fibrillation during cyclopropane anesthesia in man is, however, extremely low.

Much of the pharmacologic background on cardiac arrhythmias originated in studies of the response of the dog anesthetized with cyclopropane to the intravenous injection of epinephrine. This 'epinephrine-challenge' often produced ventricular fibrillation and the

these minimal levels of ether patients can obey commands, recognize colors and distinguish tastes. This technique deserves further study.

Ether is irritating to the respiratory tract. Its administration may be followed by increased secretions in the pharynx, trachea and bronchi, swallowing of ether-laden mucus, and by nausea and vomiting in the postoperative period. A prolonged period of induction is often observed. This is made more likely by the great solubility of ether in blood (p. 38). Attempts to hurry induction may be accompanied by cough, laryngospasm and excitement. These sequelae are not inevitable and occur less often when the drug is skillfully given.

Ether stimulates respiration (Fig. 15) through irritation of the lower respiratory tract, sensitization of pulmonary stretch receptors, stimulation of extrapulmonary sensory receptors, and possibly through the development of metabolic acidosis. The minute volume of ventilation therefore tends to remain normal or increase during an ether anesthetic, and respiratory acidosis is less likely to develop than with agents that will decrease alveolar ventilation such as cyclopropane and thiopental.

In all probability as part of a sympathetic stimulant action, ether produces bronchodilation, a characteristic which suggests its use in patients with bronchial asthma who require general anesthesia. A tendency towards mobilization of liver glycogen and a rise in blood sugar may be noted after inhalation of ether in normal man, again probably the result of sympathetic stimulation. This is of little consequence in diabetics with the methods of diabetic treatment presently available. If hypoxia and hypotension are avoided, ether causes little

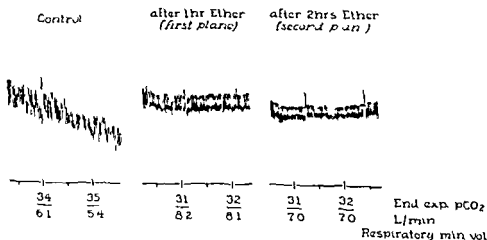


Fig. 15 The respiratory stimulant action of ether in man is indicated by an increase in respiratory rate and minute volume and a reduction in end expiratory carbon dioxide tension.

The susceptibility of patients in shock to narcosis is reflected in signs of profound overdosage if normal amounts of potent drugs are given.

Hypoxia may occur during recovery from nitrous oxide anesthesia despite regular, unobstructed breathing. This phenomenon is called diffusion anoxia, and is caused by the sudden flooding of the alveoli with large volumes of nitrous oxide which have been dissolved in the blood. It is described more fully in Chapter 23.

ETHYLENE

The background of the discovery of ethylene as an anesthetic substance is interesting. Ethylene is emitted by ripening fruit. Cut flowers if stored nearby can be harmed. This was first noted in damage to carnations made evident by a discoloration of the petals and a loss of turgor known as "sleepiness." The drug is characterized by a strong garlic-like odor which may be objectionable to patients. Ethylene is the only inhalation anesthetic agent lighter than air. This is of importance from the standpoint of sites of accumulation and possible ignition of this explosive substance. In most other respects ethylene resembles nitrous oxide, perhaps with a slightly greater potency.

ETHYL ETHER

A number of important pharmacologic observations have been made during the past two decades on the effects of ethyl ether. In common with most general anesthetics, ether depresses the isolated mammalian heart. Ether will also dilate blood vessels by a direct action upon the smooth muscle of the vessel wall. These two actions tending to produce a decrease in arterial blood pressure are counteracted by increased activity of at least certain parts of the sympathetic nervous system. So effective is this balance that blood pressure remains normal during most ether anesthetics. In the dog ether causes increased liberation of epinephrine from the adrenal medulla. This does not appear to occur in man. Rather there is a rise in the concentration of circulating norepinephrine, evidence of the increased sympathetic activity mentioned. The presumed sources of the norepinephrine are the heart muscle and the liver since adrenal medullary liberation of norepinephrine in man as the result of ether is minimal.

Recently ether analgesia has been reevaluated for clinical practice. With peripheral venous blood levels of ether as low as 2 to 29 mg. per 100 ml., reached during emergence from a greater depth of anesthesia, analgesia has been provided for the patient and adequate operative conditions for the surgeon during major operations on the heart. With

these minimal levels of ether patients can obey commands, recognize colors and distinguish tastes. This technique deserves further study.

Ether is irritating to the respiratory tract. Its administration may be followed by increased secretions in the pharynx, trachea and bronchi, swallowing of ether-laden mucus, and by nausea and vomiting in the postoperative period. A prolonged period of induction is often observed. This is made more likely by the great solubility of ether in blood (p. 38). Attempts to hurry induction may be accompanied by cough, laryngospasm and excitement. These sequelae are not inevitable and occur less often when the drug is skillfully given.

Ether stimulates respiration (Fig. 15) through irritation of the lower respiratory tract, sensitization of pulmonary stretch receptors, stimulation of extrapulmonary sensory receptors, and possibly through the development of metabolic acidosis. The minute volume of ventilation therefore tends to remain normal or increase during an ether anesthetic, and respiratory acidosis is less likely to develop than with agents that will decrease alveolar ventilation such as cyclopropane and thiopental.

In all probability as part of a sympathetic stimulant action, ether produces bronchodilation, a characteristic which suggests its use in patients with bronchial asthma who require general anesthesia. A tendency towards mobilization of liver glycogen and a rise in blood sugar may be noted after inhalation of ether in normal man, again probably the result of sympathetic stimulation. This is of little consequence in diabetics with the methods of diabetic treatment presently available. If hypoxia and hypotension are avoided, ether causes little

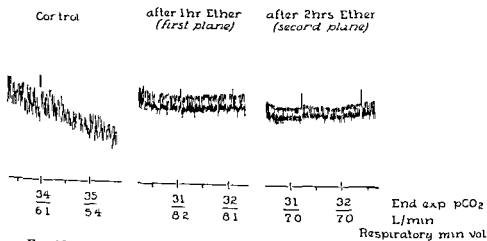


Fig. 15 The respiratory stimulant action of ether in man is indicated by an increase in respiratory rate and minute volume and a reduction in end expiratory carbon dioxide tension.

cellular damage to either the liver or kidney. Mild functional impairment of these organs may be noted for a few days after its administration.

Ether can block nerve-muscle conduction in much the same way as *d*-tubocurarine. This accounts in part for the excellent muscle relaxation provided by the drug. The neuromuscular effect is synergistic with that of *d*-tubocurarine, dimethyl *d*-tubocurarine and gallamine but not with that of decamethonium bromide or succinylcholine.

DIVINYL ETHER (VINETHENE)

In 1930 Leake reasoned that an unsaturated ether, combining the properties of ethyl ether and the unsaturated compound, ethylene, might be a useful anesthetic agent. This proved to be correct and led to the clinical use of divinyl ether, a potent, relatively non-irritating volatile liquid. Induction and emergence from anesthesia are rapid. This anesthetic is used by the open technique primarily as an induction agent to be followed by ethyl ether. It can also be used in semi-closed systems to increase the potency of nitrous oxide. It is recommended only for brief administration, since its prolonged inhalation has been followed by liver and kidney damage, despite the concomitant use of high oxygen concentrations.

In approximately 3 per cent of administrations, abnormal motor movements may be observed. These consist of twitching movements occasionally progressing to convulsions, as well as slower, less violent, rhythmic movements referred to as "running movements." The cause of these is obscure. They respond to withdrawal of the drug and inhalation of oxygen.

TRICHLORETHYLENE (TRILENE, TRIMAR)

This liquid had been used in the dry cleaning of clothing and degreasing of metals for years. Workmen leaning over vats of the substance often became intoxicated. At one time this drug was used for the treatment of tic douloureux since facial numbness followed exposure. It was not until 1934 that the drug achieved recognition as an anesthetic agent. It cannot be used in closed systems since it reacts with soda lime to form dichloroacetylene and other products which are toxic to the nervous system. Cranial nerve palsies have occurred.

Cardiac arrhythmias of the epinephrine-sensitizing type have been described. A rapid respiratory rate due to sensitization of Hering-Breuer pulmonary stretch receptors may follow its use for surgical anesthesia. Muscular relaxation is poor, largely because sufficient

depth of anesthesia is not achieved. For these reasons the drug is used for analgesia principally in obstetrics, urology and dentistry. It may be used to potentiate nitrous oxide during major operations if used in a non-rebreathing system. Special inhalers are available for its administration. Because of low volatility it is not given by the "open drop" technique. Emergence from anesthesia is usually uneventful.

ETHYL CHLORIDE

This potent, halogenated hydrocarbon is regarded by many as too dangerous for use in inhalation anesthesia. Hypotension, presumably from myocardial depression, can occur with devastating speed, serious cardiac arrhythmias have been encountered. In skillful hands, however, ethyl chloride has proved useful as an induction agent by the open technique. The onset of anesthesia occurs rapidly and with minimal excitement. Ethyl chloride has been sprayed on tissues to freeze and render them less sensitive to a surgical incision. The local anesthesia so achieved is frequently inadequate. The cooling effects of ethyl chloride on cutaneous surfaces have been used in the treatment of patients with pain, if trigger areas can be found. Here the drug is sprayed over the trigger zones and pain relief may result.

CHLOROFORM

In the United States the use of chloroform is localized to a few clinics. In the opinion of many, this volatile liquid is too dangerous for general use, despite a relatively large experience accumulated with the drug in other parts of the world. Sudden collapse of the circulation can follow small increases in inspired concentration. Cardiac arrhythmias of the epinephrine-sensitizing type can be produced experimentally. The lack of flammability of the vapor recommends it to those who fear anesthetic explosions. Until an apparatus is devised that can control the inspired concentration reliably for all who use it, it is unlikely that chloroform will enjoy a renaissance.

Search continues for better inhalation anesthetics. Xenon has been investigated as an addition to the group of gaseous agents. A potency equivalent to that of ethylene has been claimed for this substance. Fluorinated volatile liquid anesthetics are also receiving considerable attention. Trifluorethyl vinyl ether (Fluoromar) has a low flammability, causes little disturbance in cardiac rhythm, provides rapid induction and recovery and good analgesia. Tachypnea and low volatility are among its less desirable features. Bromo-chloro-trifluoroethane (Fluo-

thane) is non-explosive and potent Arterial hypotension and the development of cardiac arrhythmias appear to be its chief disadvantages

At the moment, the most commonly used and most reliable inhalation anesthetics are those to which attention was primarily directed in this chapter

REFERENCES

- Artusio, J F, Jr Ether analgesia during major surgery *J A M A* 157 33, 1955
Bunker, J P, Beecher H D Briggs, B D, Brewster, W R, and Barnes, B A
Metabolic effects of anesthesia II A comparison of acid base equilibrium in man and in dogs during ether and cyclopropane anesthesia *J Pharmacol & Exper Therap* 102 62, 1951
Dripps R D The immediate decrease in blood pressure seen at the conclusion of cyclopropane anesthesia cyclopropane shock *Anesthesiology*, 8 15, 1947
Dripps R D, and Severinghaus J W General anesthesia and respiration *Physiol Rev* 35 741 1955
Martin, S J, and Rovenstine, E A Vinethene Recent laboratory and clinical evaluation *Anesthesiology* 2 285, 1941
Papper E M Renal function during general anesthesia and operation *J A M A* 152 1686 1953
Price, H L Conner, E H and Dripps, R D Concerning the increase in central venous pressure and arterial blood pressures during cyclopropane anesthesia in man *Anesthesiology* 14 1 1953
Waters R M Chloroform A Study after 100 Years University of Wisconsin Press Madison, 1951

"Open Drop" Anesthesia

THE administration of volatile liquid anesthetics by the "open drop" technique continues to be of use despite the development of more refined methods. It is of value to the occasional anesthetist for it can be applied to almost any patient requiring anesthesia. It is useful in the anesthetic management of infants and small children in whom closed system techniques may be troublesome because of the increased resistance to breathing and the dead space within the apparatus. If understood, it is relatively safe, simple, and requires little equipment.

PREPARATION

This is similar to that for any inhalational anesthetic. When children under five years of age are anesthetized by the open drop technique, narcotics should be omitted from the preanesthetic medication. The respiratory tidal volume of the child is small, and respiratory depression from narcotics in decreasing pulmonary exchange may lead to hypercapnia and hypoxia. Unfortunately, there is no way to augment respiration with this technique.

The equipment consists of a mask covered with gauze eight individual layers thick. Additional gauze should be available to increase the dead space temporarily, when placed around the mask, or to replace the mask gauze if it becomes wet. Oropharyngeal airways, suction catheters, oxygen, and a mask and bag for resuscitation complete the equipment. Diethyl ether (Vinethene), ethyl chloride or diethyl ether should be available as needed. After removal of the soft metal cap of the ether container, a cork with two deep wedges is inserted. A long gauze wick is placed in one of the wedges (Fig. 17, C).

Care must be exercised to avoid injury to the patient's eyes. Gauze sponges may brush the cornea if the eyes open during anesthesia. At

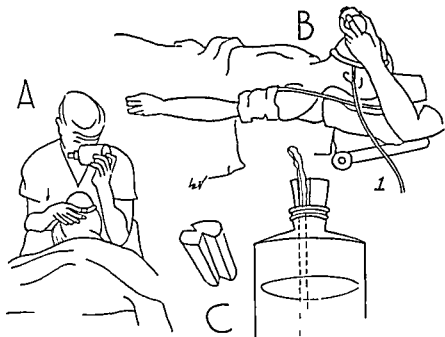


Fig 17 Technique of open drop anesthesia A Method of supporting the head with the arms B Lateral view showing support of head and insufflation of oxygen (1) beneath the mask C Cork cut and wick in place to drip ether

the beginning of induction it is best not to cover the eyes in children as they may become frightened. After anesthesia has been induced, the eyes can be protected with a piece of rubber dam, the instillation of boric acid ointment, or by taping the eyes shut. The last is probably preferable. Although diethyl ether is often accused of producing ulceration of the conjunctiva or cornea, rarely is this true. Ulceration is more likely to result from direct trauma.

INDUCTION

The anesthetist should sit directly behind the patient with the stool adjusted so that he may look down on the patient's face. The mask should be held with one hand and the opposite forearm or elbow should be resting next to the patient's head (Fig 17, A). If struggling occurs, one is in a position to steady the patient's head between the hand and the elbow and forearm. Induction can be long and stormy. An assistant is needed in this situation more than any other in anesthesia.

In children, distraction is essential. Soothing talk about school, playmates or television is helpful. Games of counting and other diversionary practices assist in a smooth induction. Truth, demonstration and gentleness produce better results than falsehoods and force. In

adults one should engage in a description of the sensations of induction just as with other techniques of general anesthesia

One should drip the anesthetic slowly at first, then increase rapidly to tolerance. The mistake of the beginner is to reverse this procedure, that is, too great a concentration is given during the first few seconds and too little during the remainder of the induction. The latter results in an induction that makes little progress—the patient coughs, he neither awakens completely, nor does he progress into surgical anesthesia. It is common at this point to encounter profuse mucous and salivary secretions. Laryngeal irritation, vocal cord spasm, partial respiratory obstruction, retching or vomiting may follow. It becomes exceedingly difficult to reach a deeper plane of anesthesia.

To avoid some of these difficulties, divinyl ether or ethyl chloride can be used for a rapid induction of anesthesia. These drugs should be administered until the patient loses consciousness and smooth regular respirations have become established. Diethyl ether can then be substituted for the induction agent, or, if the operation is to be a brief one, divinyl ether can be continued. When ether is substituted, the initial flow should be slow and then rapidly increased. It is unwise to use ethyl chloride for more than two or three minutes since prolonged administration may lead to cardiovascular collapse. To increase the speed of induction, gauze pads may be applied to the sides of the mask to produce hyperventilation by temporarily increasing the inspiratory carbon dioxide tension. When induction is prolonged it may be necessary to change the gauze, for a mask made cold by the vaporization of ether may condense the moisture of expired air. This in turn further retards vaporization of the ether.

As soon as possible, an oxygen delivery tube or ether hook is placed under the mask or into the mouth. If room air is breathed during the 'open drop' technique, the inspired oxygen concentration will be below normal and a lowered oxygen saturation of hemoglobin in arterial blood will result. There is a rough inverse relationship between the partial pressure of ether vapor and oxygen under the mask—the higher the ether pressure the lower the oxygen. In addition to dilution of oxygen by ether vapor, there is dilution by water vapor and by carbon dioxide that may accumulate because of the increased dead space created by the gauze. This oxygen deficit can and should be corrected by the addition of a 500 ml per minute flow of oxygen (Fig 17, B).

Another important aspect of the 'open drop' technique is the effect of mask temperature on the vaporization of liquid ether. Thermocouples placed between the gauze layers indicate that the temperature is quickly lowered to from -2 to -8°C . This places an upper limit of 170 or

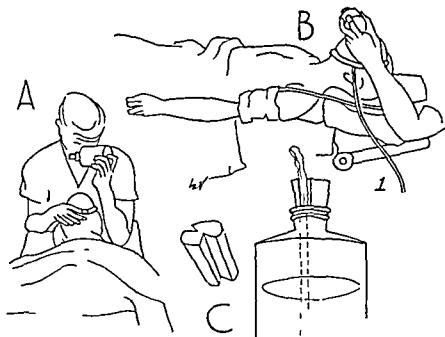


Fig 17 Technique of open drop anesthesia A Method of supporting the head with the arms B Lateral view showing support of head and insufflation of oxygen (1) beneath the mask C Cork cut and wick in place to drip ether

the beginning of induction it is best not to cover the eyes in children as they may become frightened. After anesthesia has been induced, the eyes can be protected with a piece of rubber dam, the instillation of boric acid ointment, or by taping the eyes shut. The last is probably preferable. Although diethyl ether is often accused of producing ulceration of the conjunctiva or cornea, rarely is this true. Ulceration is more likely to result from direct trauma.

INDUCTION

The anesthetist should sit directly behind the patient with the stool adjusted so that he may look down on the patient's face. The mask should be held with one hand and the opposite forearm or elbow should be resting next to the patient's head (Fig 17, A). If struggling occurs, one is in a position to steady the patient's head between the hand and the elbow and forearm. Induction can be long and stormy. An assistant is needed in this situation more than any other in anesthesia.

In children, distraction is essential. Soothing talk about school playmates or television is helpful. Games of counting and other diversionary practices assist in a smooth induction. Truth, demonstration and gentleness produce better results than falsehoods and force. In

Fundamentals of Muscle Relaxants

PRIOR to 1940 curare was a medical curiosity. There were no pure preparations. Injection of crude solutions into animals resulted in such depression of blood pressure that the possibility of use in man seemed remote. Textbooks limited discussion of curare to a few paragraphs. With the introduction of *d*-tubocurarine in pure form its use for muscle relaxation increased rapidly. Before this time abdominal muscular relaxation for surgical operations, and relaxation of the jaw and larynx for tracheal intubation, were achieved through deep general anesthesia. This degree of anesthesia was accompanied by many side effects and was followed by a postoperative course characterized by 'hangover'. With use of the muscle relaxants, less general anesthesia is required. One can therefore ask, 'Do the hazards of deep general anesthesia outweigh those associated with the administration of muscle relaxants?'

Before this question can be approached—and it cannot really be answered with certainty—one should know something about the pharmacological properties of the commonly used muscle relaxants.

ACTION

The most important action of these drugs is to interfere with the passage of impulses from a motor nerve to skeletal muscle. This interference takes place at the nerve-muscle junction, a zone composed of motor nerve terminals and a specialized section of the muscle cell membrane called the end plate. Although there is anatomic discontinuity between nerve and muscle, the gap in transmission is bridged by

125 mm Hg on the vapor pressure of ether under ideal conditions. In clinical practice unless the mask is warmed, it is difficult to exceed 100 mm Hg or about 13 per cent. Closed system techniques do not have this degree of limitation.

Common sense is a part of any technique. Too often this is lacking during the conduct of "open drop" anesthesia. The following are the more frequent errors: (1) Pouring ether onto a mask which is already saturated. Obviously the drug must be vaporized before it can be inspired. One cannot increase the concentration of anesthetic in the inspired air by supersaturation. (2) Continuing to administer an anesthetic when the patient is not breathing. Breath-holding may be a manifestation of the excitement stage, it may be produced reflexly by irritation of the respiratory tract, or it can be related to a high cerebral arterial concentration of the anesthetic. If the anesthetic is poured on the mask during periods of apnea, the whole cycle of apnea may be repeated when breathing resumes. (3) Failure to take advantage of the hyperpnea. After breath-holding or following struggling and excitement, respirations are often full and deep due to accumulated carbon dioxide. This increased ventilation permits one to raise the anesthetic concentration and to carry the anesthetic agent into the blood stream more rapidly.

MAINTENANCE

Anesthesia is best maintained by a steady drop of anesthetic agent, though the rate may vary, depending upon the phase of anesthesia. Thus, the rate is slowed at the beginning of anesthesia, increases steadily until the desired plane of anesthesia is reached, and then slowed during the maintenance. The longer the anesthesia continues, the slower the rate of administration. If the rate of drip is irregular, the anesthesia will vary from light to deep, depending upon the concentration beneath the mask.

The pulse may be counted by a finger of the left hand making contact with the temporal artery or an artery over the ramus of the jaw. It may be necessary to interrupt the administration of anesthetic while the pulse is counted, and also when the blood pressure is recorded.

APPRAISAL

There is a self-contained art in the administration of anesthesia by the "open drop" technique. It can be performed with satisfaction to patient, anesthetist and surgeon alike. Those strongest in their condemnation of the technique are usually those least able to use it correctly.

curarine, which depresses transmission at synapses in the sympathetic ganglia and may produce a decreased contractility of blood vessels that are under sympathetic control. Gallamine depresses transmission through cardiac vagal ganglia and tachycardia results. Both drugs are alleged to minimize the reflex circulatory and respiratory effects of intra-abdominal or intrathoracic manipulation although the evidence is not convincing. Succinylcholine may have a mild sympathetic ganglionic stimulant property, an effect partially responsible for a rise in blood pressure sometimes observed.

There is no unanimity of opinion as to a central nervous system action of the muscle relaxants. Some believe that synaptic transmission in the brain is depressed, although a clinically important action has not been obvious.

Succinylcholine crosses the placental barrier. There are fragmentary data that suggest that other relaxants do not share this property.

METABOLISM AND EXCRETION

Intelligent use of any drug requires knowledge of its fate in the body. Decamethonium is almost completely eliminated by the kidneys. Impaired renal function may therefore be associated with accumulation of this substance. Since 75 to 90 per cent of gallamine is similarly excreted the same thesis applies. Approximately 60 per cent of dimethyl *d*-tubocurarine, 35 per cent of *d*-tubocurarine and only 5 to 10 per cent of succinylcholine appear in the urine.

Succinylcholine is hydrolyzed rapidly in the plasma, chiefly by non-specific plasma cholinesterase. Enzymatic hydrolysis takes place in two stages. The drug is rapidly changed to succinylmonocholine, which in turn is broken down more slowly to choline and succinate. In the presence of severe liver disease and malnutrition, or if, for other as yet poorly understood reasons, the concentration of plasma cholinesterase in the blood is low, the action of succinylcholine may be abnormally prolonged. It must, however, be emphasized that abnormal prolongation of succinylcholine effects occurs in the presence of normal amounts of the enzyme. Other factors must therefore be involved. Of possible significance in association with the prolonged effect of succinylcholine is the fact that the mono-ester has a blocking action of its own.

Following the intravenous injection of *d*-tubocurarine or dimethyl *d*-tubocurarine, 60 to 90 per cent of the injected drug disappears from the blood stream within a few minutes (Fig. 18). This rapid decline in blood stream concentration is due to distribution of these substances throughout body fluids and tissues, and does not signify prompt

the chemical transmitting agent, acetylcholine. As the result of motor nerve activity acetylcholine is released from the nerve terminals at the cell membrane of muscle. A phenomenon known as depolarization accompanies this, an electrical potential develops at the end-plate. When this reaches a certain magnitude a wave of depolarization in the muscle cell is produced and the muscle contracts. Acetylcholine is destroyed in a fraction of a second by the enzyme cholinesterase, and a restorative process begins, culminating in the return of the muscle to its precontractile state.

This natural process can be interrupted by preventing acetylcholine from reaching the receptor area of the muscle cell membrane. *d*-Tubocurarine is believed to produce muscular paralysis in this way. The hypothesis is *d*-tubocurarine blocks the action of the chemical transmitter through combination with receptors usually occupied by acetylcholine. Gallamine (Flaxedil) and dimethyl *d*-tubocurarine (Metubine) act in similar fashion.

Other drugs also interfere with transmission at the nerve-muscle junction and produce muscular paralysis thereby. Two commonly used synthetic substances are decamethonium (Syncurine) and succinylcholine (Anectine, Scoline, Quelcin). The mode of action of these two compounds has not yet been agreed upon. Formerly it was believed that prolonged depolarization of the end-plate region was entirely responsible for the block. More recent work indicates that depolarization does occur, but that a defect in conduction persists long after depolarization has disappeared. The block then assumes some of the characteristics of that produced by *d*-tubocurarine. It is tempting to predict that ultimately we shall find a fundamental similarity in the action of the two groups of relaxants.

The doses of muscle relaxants used intravenously in anesthesia may be compared as follows:

	COMPARABLE CLINICAL DOSES (mg)	ONSET OF ACTION (seconds)	DURATION OF ACTION (minutes)
Decamethonium	3	120-180	15-20
Dimethyl <i>d</i> tubocurarine	4-5	90-150	25
<i>d</i> -Tubocurarine	18	90-150	30
Succinylcholine	30	30-75	4-6
Gallamine ^a	50	90-120	15-20

In addition to an action at the nerve-muscle junction, the muscle relaxants can reduce transmission of impulses through ganglia of the autonomic nervous system. This has been demonstrated for *d*-tubo-

laxants This reversal is most likely to occur when *d*-tubocurarine, dimethyl *d*-tubocurarine or gallamine has been used Presumably an anticholinesterase agent, by preventing the destruction of acetylcholine, permits this chemical transmitter to accumulate and to restore neuromuscular conduction An antagonism has also been demonstrated clinically and experimentally for decamethonium and succinylcholine This suggests that *prolonged depolarization is not the factor involved* in the continuing action of these two muscle relaxants and supports the idea that the basic effect of all of the substances is similar

If *d*-tubocurarine is injected and its effect allowed to wear off partially, the subsequent injection of decamethonium or succinylcholine in the usual paralyzing doses is relatively ineffective The explanation for this antagonism is puzzling

SYNERGISM

d-Tubocurarine acts synergistically with ether, so that smaller amounts of the former are needed to provide muscular relaxation during ether anesthesia Ether can be shown to have a blocking action at the nerve-muscle junction, a property not shared by thiopental and cyclopropane except with excessive doses Ether synergism with *d*-tubocurarine (and probably dimethyl *d*-tubocurarine and gallamine) thus seems to represent the additive effect of two drugs both acting to decrease transmission from nerve to muscle Other volatile anesthetics should be studied for this effect It has also been observed clinically that if large amounts of succinylcholine have been given as with the continuous intravenous infusion of an 0.1 per cent solution, the subsequent administration of 12 to 15 mg of *d*-tubocurarine may produce a profound paralyzing effect

EFFECT ON THE RESPIRATORY SYSTEM

All of the muscle relaxants can reduce the minute volume of respiration or produce apnea through neuromuscular blockage At the end of an operation this effect should have disappeared Unfortunately, this does not always happen, and inadequate ventilation or apnea may persist into the postoperative period for as long as one to six hours The particular relaxant given does not seem to be important This undesirable effect is noted more often in patients whose preoperative physical condition is poor

If *apnea* is present at the end of an operation at least three different factors may be involved, each of which demands different treatment

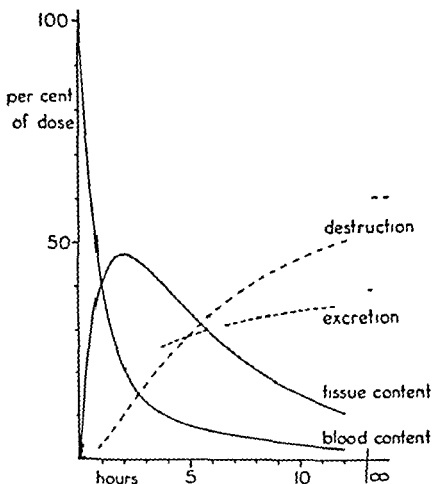


Fig 18 Distribution of *d* tubocurarine in man after rapid intravenous injection. Theoretical curves calculated from urinary excretion rates (From Kalow W J Pharmacol & Exper Therap vol 109 1953)

destruction. Depots can become saturated if large amounts are given, and prolonged paralytic effects may be observed. Under these circumstances the factors in metabolism—liver conjugation and kidney excretion—become important. Since extravascular compartments can hold about half the given dose of *d*-tubocurarine, a considerable amount can be stored temporarily following two or three intravenous injections within the space of an hour. If dehydration, electrolyte or fluid shifts develop, delayed mobilization of *d*-tubocurarine may cause a return of paralysis after drug action had apparently ceased.

ANTAGONISM

Neostigmine and edrophonium (Tensilon), anticholinesterase drugs, may reverse the neuromuscular block produced by some of the re-

laxants This reversal is most likely to occur when *d* tubocurarine, dimethyl *d* tubocurarine or gallamine has been used Presumably an anticholinesterase agent, by preventing the destruction of acetylcholine, permits this chemical transmitter to accumulate and to restore neuromuscular conduction An antagonism has also been demonstrated clinically and experimentally for decamethonium and succinylcholine This suggests that prolonged depolarization is not the factor involved in the continuing action of these two muscle relaxants and supports the idea that the basic effect of all of the substances is similar

If *d* tubocurarine is injected and its effect allowed to wear off partially, the subsequent injection of decamethonium or succinylcholine in the usual paralyzing doses is relatively ineffective The explanation for this antagonism is puzzling

SYNERGISM

d Tubocurarine acts synergistically with ether, so that smaller amounts of the former are needed to provide muscular relaxation during ether anesthesia Ether can be shown to have a blocking action at the nerve muscle junction, a property not shared by thiopental and cyclopropane except with excessive doses Ether synergism with *d*-tubocurarine (and probably dimethyl *d* tubocurarine and gallamine) thus seems to represent the additive effect of two drugs both acting to decrease transmission from nerve to muscle Other volatile anesthetics should be studied for this effect It has also been observed clinically that if large amounts of succinylcholine have been given, as with the continuous intravenous infusion of an 0.1 per cent solution, the subsequent administration of 12 to 15 mg of *d* tubocurarine may produce a profound paralyzing effect

EFFECT ON THE RESPIRATORY SYSTEM

All of the muscle relaxants can reduce the minute volume of respiration or produce apnea through neuromuscular blockage At the end of an operation this effect should have disappeared Unfortunately, this does not always happen, and inadequate ventilation or apnea may persist into the postoperative period for as long as one to six hours The particular relaxant given does not seem to be important This undesirable effect is noted more often in patients whose preoperative physical condition is poor

If apnea is present at the end of an operation at least three different factors may be involved, each of which demands different treatment

One must consider the effects of (1) controlled respiration, (2) an endotracheal tube, (3) the muscle relaxant *per se*

Controlled Respiration Excessive depth and rate of artificial ventilation, whether provided by manual compression of the breathing bag or by machine, can reduce arterial carbon dioxide tension to a point where spontaneous respiration ceases. The pressure used for inflation of the lungs may stimulate Hering-Breuer stretch receptors which exert an inhibitory effect on the respiratory center via the vagus nerves. The combination of these factors might result in an apnea which could be erroneously interpreted as being due to muscle relaxants. Treatment consists of interrupting pulmonary ventilation so that carbon dioxide concentration in arterial blood is restored to normal, and the rhythmic discharge of the respiratory center (decreased by low carbon dioxide and inhibitory reflexes) is reestablished. One should not cease ventilating the lungs for a period long enough for hypoxia to develop.

Endotracheal Tube The presence of this foreign body in the trachea, particularly with an inflated cuff, may cause apnea. The reflex pathway for this is not clear, but deflation of the cuff, or withdrawal of the tube, may remove an inhibitory stimulus and permit resumption of spontaneous breathing.

Muscle Relaxant Action per se Overdosage with the muscle relaxant may cause prolonged apnea either by saturating tissue and fluid depots, or by the overwhelming of detoxifying and metabolic mechanisms. The latter may be deficient through disease of such organs as the liver or kidney. Electrolyte imbalance, particularly that of low plasma potassium, can contribute to prolonged neuromuscular block.

It is safest to use the muscle relaxants over long periods of time, without stopping respiration completely, so that there is always some degree of spontaneous respiration to guide the anesthetist. Should respiratory depression or apnea persist at the end of operation, assistance to breathing, or artificial ventilation, is the basic element in treatment. If anticholinesterases are used (neostigmine 1 to 2 mg, edrophonium 10 mg intravenously) to antagonize the competitive neuromuscular type of drug, they should be preceded by 0.4 to 0.6 mg atropine sulfate intravenously to minimize the increase in respiratory tract secretions and other parasympathetic effects. If correction of potassium deficiency is attempted with an intravenous solution, electrocardiographic guidance provides a safeguard against the development of cardiac arrhythmias or asystole. The use of calcium salts as antagonists to succinylcholine has also been suggested. In general,

however, it seems best to rely on adequate pulmonary ventilation and passage of time

EFFECT ON THE CIRCULATORY SYSTEM

As one reviews reports of death following the use of the muscle relaxants, it is evident that in addition to respiratory abnormalities, arterial hypotension frequently occurs. One cause for circulatory depression is the use of excessive positive pressure to inflate the lungs. Reduction of arterial pressure consequent to raised airway pressure is a well known response. A second possibility lies in the combined use of intravenous thiopental and relaxants. Thiopental *per se* can cause a decrease in arterial pressure. Some relaxants cause release of tissue histamine, a substance predisposing to hypotension. Some relaxants block sympathetic ganglionic transmission and interfere with vasomotor reactivity in this way. Hypotension must be treated promptly by the measures suggested in Chapter 20.

APPRAISAL

The muscle relaxants are powerful drugs and have unquestioned potential for harm. Their use has been followed by death. On the debit side of the ledger is the fact that a considerable degree of knowledge and skill is required for their safe administration. On the credit side is the lesser depth of anesthesia needed, and a possible reduction in untoward reflexes associated with surgical manipulation. In our opinion, the balance cannot yet be struck.

REFERENCES

- Foldes, F. F. Muscle Relaxants in Anesthesiology. Charles C Thomas, Springfield, Ill., 1957.

Intubation of the Trachea

THE advantages of intubation of the trachea are unquestioned. Following tracheal intubation, respiratory dead space is reduced, control of respiration is facilitated, and the removal of secretions from the tracheobronchial tree simplified. The patient can be placed in any position for operation without compromising the airway. The anesthetist can be at some distance from the head of the patient, yet retain adequate control of the anesthetic.

Tracheal intubation has become commonplace and the technique is often used for the convenience of the anesthetist rather than for the good of the patient. There appears to be a belief among anesthetists that there are no untoward sequelae of endotracheal anesthesia. For this reason emphasis will be placed on complications arising as a result of the use of the laryngoscope and endotracheal tubes. All of the following complications have been seen by the authors.

Immediate Traumatic Complications

- 1 Cut, bruised or swollen lips and tongue
- 2 Chipped, loosened or dislodged teeth
- 3 Laceration or hemorrhage in the pharyngeal mucous membrane
- 4 Dislodgement of adenoidal tissue
- 5 Submucosal hemorrhage of the vocal cords

Technical Complications

- 1 Increased resistance to breathing imposed by tubes (adapters or connectors) with an inside diameter too small
- 2 Obstruction of the lumen of the endotracheal tube due to collapse or kinking. Obstruction may also occur if a patient bites on the tube or when a cuff has been overinflated thereby collapsing the

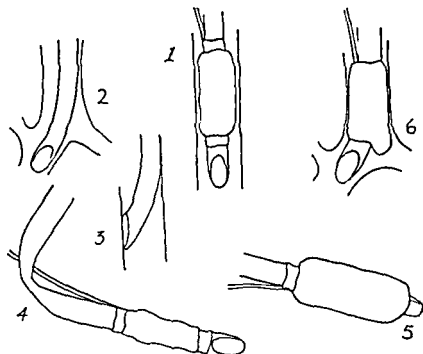


Fig 19 Accidents with tracheal intubation 1 Normal position of the tube 2 Intubation of the right main bronchus 3 Tube opening against side of trachea with one way valve effect 4 Kinked tube 5 Endotracheal cuff inflated with partial occlusion of opening 6 Inflated cuff partially occluding left main bronchus

wall of the tube, when the bevel of the tube impinges upon the tracheal wall and acts as a one-way valve, when the cuff overrides the tube's opening, there may be obstruction within the tube due to dried secretions (Fig 19)

- 3 Unrecognized esophageal intubation
- 4 *Endobronchial intubation due to the use of a long endotracheal tube* Usually the right main stem bronchus is intubated, leaving the left lung unaerated. Bronchial obstruction from the cuff (Fig 19)
- 5 Dislodgment of tube because of failure to anchor it properly. Displacement into the pharynx or further insertion into a bronchus can occur

Undesirable Circulatory and Respiratory Effects

- 1 Transient hypertension and tachycardia can follow tracheal intubation when accomplished without adequate topical or general anesthesia
- 2 Conversely, hypotension may be associated with tracheal in-

Intubation of the Trachea

THE advantages of intubation of the trachea are unquestioned. Following tracheal intubation, respiratory dead space is reduced, control of respiration is facilitated, and the removal of secretions from the tracheobronchial tree simplified. The patient can be placed in any position for operation without compromising the airway. The anesthetist can be at some distance from the head of the patient, yet retain adequate control of the anesthetic.

Tracheal intubation has become commonplace and the technique is often used for the convenience of the anesthetist rather than for the good of the patient. There appears to be a belief among anesthetists that there are no untoward sequelae of endotracheal anesthesia. For this reason emphasis will be placed on complications arising as a result of the use of the laryngoscope and endotracheal tubes. All of the following complications have been seen by the authors.

Immediate Traumatic Complications

- 1 Cut, bruised or swollen lips and tongue
- 2 Chipped, loosened or dislodged teeth
- 3 Laceration or hemorrhage in the pharyngeal mucous membrane
- 4 Dislodgement of adenoidal tissue
- 5 Submucosal hemorrhage of the vocal cords

Technical Complications

- 1 Increased resistance to breathing imposed by tubes (adapters or connectors) with an inside diameter too small
- 2 Obstruction of the lumen of the endotracheal tube due to collapse or kinking. Obstruction may also occur if a patient bites on the tube or when a cuff has been overinflated thereby collapsing the

should be inspected before induction of anesthesia. One may detect loose, broken or diseased teeth, dental plates or loose appliances, and occasionally chewing gum. Gentleness is the keyword in tracheal intubation. The maneuver should not be hurried or forceful nor should the anesthetist's attention be so fixed at the tip of the laryngoscope that he disregards other structures of the mouth and pharynx.

The student must first become acquainted with the anatomy of the pharynx, larynx and trachea. An experienced anesthetist must be in attendance during the student's early experiences with tracheal intubation. Finally, it is worth while for the student to describe aloud the anatomic structures as he views them. There is much to recommend intubation in conscious patients, under topical anesthesia, for this obviates the need for haste and places a premium on gentleness. The next best alternative is in the patient deeply anesthetized with ether. Speed again is not essential, for the patient continues to breathe and remains relaxed.

In our experience the most common cause for failure to insert an endotracheal tube is inadequate depth of anesthesia. The lack of re-

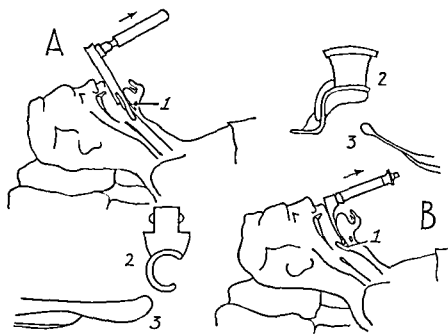


Fig 20 Upward lift of laryngoscope at 45 degree angle to expose glottis. A. Straight blade beneath the (1) epiglottis 2 End view of straight blade with semicircular construction to keep tongue to the left 3 Tip of straight blade to lift hyoid bone when placed beneath epiglottis B Curved blade proximal to (1) epiglottis 2 End view of curved blade with right angle construction to keep tongue to left and flange to lift tongue 3 Tip of curved blade

tubation secondarily. This is the result of increased airway pressure, precipitated by coughing and bucking, or by attempts to ventilate the lungs during chest wall spasm. The circulatory effects of raised airway pressure are amplified in Chapter 20.

- 3 Hypoxia or hypercapnia. These sequelae will arise if too much time is taken to insert the tube or may follow the responses noted in the preceding paragraph.
- 4 Increased resistance to respiration as noted under Technical Complications. The patient may respond by developing hypercapnia, hypoxia, hypertension and tachycardia.

Complications Following Tracheal Intubation

- 1 Tracheal edema and infection from improperly sterilized equipment or contaminated lubricants.
- 2 Pulmonary infection as a result of passage of endotracheal tube through a contaminated area (through the nasopharynx in the presence of acute sinusitis) and transmission of pathogenic bacteria into lungs.
- 3 Tracheal and laryngeal edema indicative of an allergic response to the material incorporated in the endotracheal tube or the lubricant.
- 4 Postintubation ulceration and granulomata of the vocal cord from irritation or trauma by the tube.
- 5 Hoarseness, probably due to a mild laryngitis from the presence of a foreign body in the glottis.
- 6 Ulceration of the tracheal mucous membrane.

This list of complications is impressive. It is not meant to dissuade the anesthetist but rather to remind him of what may ensue with a careless technique. These sequelae occur more commonly during the training period of the anesthetist and become less frequent as skill develops. A critical analysis of one's own technique as well as that of others will do much to increase the safety and comfort of the patient as far as tracheal intubation is concerned.

TECHNIQUES OF TRACHEAL INTUBATION

Before discussing the details of the techniques of tracheal intubation there are certain general principles to be stressed. Endotracheal tubes of the proper size and length should be selected. It is wise to have tubes of more than one size ready for use. One should look through the lumina of tubes prior to insertion into the trachea. This assures patency and freedom from foreign material. *The patient's mouth*

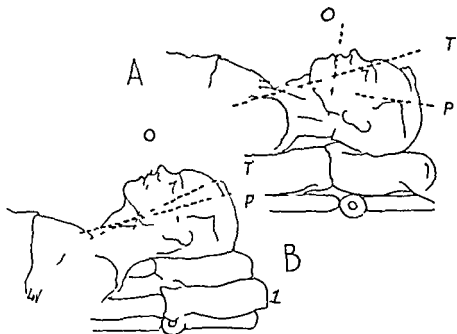


Fig 21 Position of the head for laryngoscopy and intubation of the trachea A Ordinary position T Axis of the trachea P Axis of the pharynx O Axis of the oral cavity B Modified position achieved with an extra head rest Flexion of the cervical spine and extension at the atlanto-occipital joint bring the three axes more nearly into line

of the glottis is certain Rarely an attempt to intubate must be made when the glottic opening cannot be brought into view Under these circumstances one can identify the epiglottis and possibly the arytenoid cartilages before advancing the tube Endotracheal tubes are introduced to one side of the laryngoscope, leaving the lumen free for vision

Oral Visual Intubation (Curved Blade)

In general the technique is the same as above except that exposure of the glottic opening is not obtained through elevation of the laryngoscope blade placed under the laryngeal surface of the epiglottis In this case the tip of the curved blade remains in contact with the tongue and rests in the area between the epiglottis and base of the tongue Elevation of the laryngoscope causes retraction of the epiglottis toward the blade and exposure of the glottic opening by the pull on the glosso-epiglottic ligament If the tip of the blade is inserted too deeply, retraction of the epiglottis cannot occur and exposure becomes difficult

The theoretical advantages of a curved blade are derived from the fact that the sensory innervation of the laryngeal surface of the epi-

laxation also leads to a higher incidence of trauma. On the other hand, the most common complication encountered during insertion of an endotracheal tube with the aid of a muscle relaxant is hypoxia. The relaxation provides a false sense of security, and too long a time is taken for intubation. As the relaxant effect disappears, coughing and bucking occur, since the depth of anesthesia is inadequate.

A laryngoscope is essentially an illuminated tongue blade. The handle of the instrument is a battery case. There are many styles of laryngoscopic blades, but all fall into two main categories, the straight and the curved (Fig. 20). Both are usually L shaped in cross section, the purpose of the vertical projection being to keep the tongue from the field of vision. Since the techniques employed with each differ in some measure, they will be discussed separately. We have advocated that the same laryngoscope be used until familiarity with its use has developed. Uncuffed tubes are preferable for first attempts as they are easier to insert.

Oral Visual Intubation (Straight Blade)

The head should be in the midline and the occiput supported upon a small pillow in order to bring the axis of the trachea, pharynx and mouth into line (Fig. 21). Protective lead or adhesive tape shields on the teeth may be used, but emphasis should be placed on attention to technical details and gentleness to prevent injury to the teeth and lips. It is well to moisten the blade of the laryngoscope with water or lubricant to facilitate passage over dry mucous membranes.

The laryngoscope blade should be held in the left hand with the thumb and fingers of the right separating the lips and teeth and preventing the lips from being caught between blade and teeth. The blade should be inserted on the right side of the mouth and moved toward the center, displacing the tongue to the left. The teeth or gums should not be used for leverage but exposure should be gained by an upward lift of the instrument in a 45 degree angle from the face (Fig. 20).

When the epiglottis is exposed, the blade should be advanced a few millimeters beneath the tip of the structure. An upward and outward lift follows. If the head is now extended with the right hand under the occiput the glottis should come into view. Overinsertion of the laryngoscope blade results in elevation of the larynx as a whole rather than exposure of the glottic opening. The blade may expose the esophagus which is recognized by its oval opening and lack of arytenoid cartilages.

Intubation of the trachea should only be attempted when visualization

INTUBATION OF THE TRACHIL

presence of acute sinusitis and mastoiditis, since pathogenic bacteria may be carried into the trachea

Nasotracheal tubes must be soft so as not to injure the nasal mucous membrane or turbinates, yet must be of the proper consistency to resist compression and maintain a reasonable curve. The tube must be well lubricated to minimize trauma. A laryngoscope and laryngeal forceps must be at hand should blind intubation fail. Attempts at blind intubation should be abandoned if not immediately successful and the tube then inserted under direct vision.

The patient's occiput rests upon a small pillow with the chin extended. The tube should be introduced with the concavity forward in the sagittal plane. Advancement of the tube must be slow and gentle. Roughness, thick rigid tubes, poor lubrication, and forcing an obstruction will cause nasal hemorrhage. A combination of looking, listening to breath sounds, and sensing with the fingers contributes to successful blind nasal intubation. One observes the neck in the region of the hypopharynx and larynx for lateral bulging caused by the tip of the advancing tube, one listens for maximal breath sounds, and one senses resistance to the tube in its passage. If hyperpnea can be induced during these maneuvers, it is helpful. The vocal cords will be maximally abducted with inspiration and the patient almost appears to inhale the tube. Entrance of the tube into the larynx is often accompanied by a cough. The position of the tube can be tested by connecting the tube to the rebreathing system and attempting to expand the lungs.

Extubation

The removal of the endotracheal tube at the conclusion of anesthesia may result in undesirable sequelae. While it is essential to rid the trachea and pharynx of secretions prior to extubation, one should not persist in such attempts to the point of continued coughing and cyanosis. The aspirating catheter should be passed quickly through the endotracheal tube, the secretions aspirated, and the catheter withdrawn. After aspiration the patient should be given oxygen to breathe, following which the endotracheal tube is withdrawn. The withdrawal should be reasonably quick if an aspirating catheter is still in the tube, for if the catheter is allowed to remain in contact with the cords, hemorrhage or severe laryngospasm will occur. After withdrawal the patient should again be given oxygen to breathe. In children, it is best not to withdraw the tube with the catheter inside because of the high incidence of laryngospasm. An endotracheal tube should not be withdrawn in a cyanotic patient. If laryngospasm occurs, the hypoxia may worsen and cardiac arrest may

glottis arises from the superior laryngeal branch of the vagus nerve. Stimulation of this surface by the instrument may lead to reflex vocal cord spasm and coughing. Conversely innervation of the pharyngeal surface is derived from the glossopharyngeal nerve. Stimulation of this surface is less likely to cause laryngeal spasm. The curved blade allows more room for the passage of the tube than does the average straight blade. Sometimes exposure of the glottis is not as good as that obtained with the straight blade and a stylet must be used in a higher proportion of cases. In short, thick-necked individuals and those with a more superiorly situated larynx, the curved blade is often quite useful.

Orotracheal Intubation in a Conscious Patient

An anesthetist should learn to intubate the trachea with the patient awake. In order to do this, topical anesthetization is accomplished by spray, by application of the local anesthetic with cotton swabs, or by transtracheal injection of the anesthetic. The application can be performed under direct vision (with laryngoscope) or indirect vision (reflected light and laryngeal mirror). Structures should be anesthetized in the following order: base of tongue, epiglottis, oropharynx, pyriform fossae, vocal cords, and finally, larynx and upper trachea by instillation through the glottis. A minimal amount of solution should be used to prevent reactions (not more than 2 to 3 cc. of 10 per cent cocaine for the entire procedure).

Orotracheal intubation with the patient awake can be used to advantage in the following situations: tumor of the neck or upper mediastinum displacing the trachea or larynx, tumor or inflammatory swelling encroaching upon the mouth or pharynx, malformations of the jaws, and in high acute intestinal obstruction wherein the hazard of aspiration of vomitus during induction of anesthesia is great. In the latter case, a cuffed tube should be used and the cuff inflated prior to induction of general anesthesia.

Blind Nasotracheal Intubation

Many anesthetists neglect this valuable technique. Blind nasotracheal intubation may be the method of choice for oral surgery and maxillofacial surgery. It is invaluable in certain emergency situations where an airway is needed quickly, such as in the obese, full-necked person who obstructs easily or in the patient with hypoglossal, pharyngeal or cervical edema. It is also indicated in patients with fractured jaws. It should be avoided in patients with fracture of the nose or in the

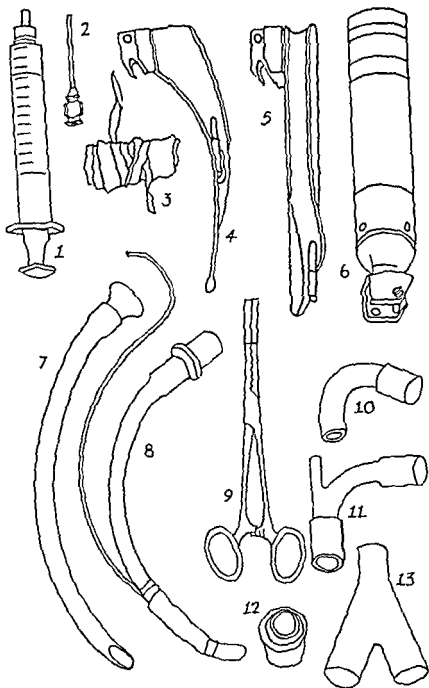


Fig 22 Endotracheal equipment 1 Syringe and (2) blunt tipped needle to inflate cuff on endotracheal tube 3 Bite block 4 Curved laryngoscope blade 5 Straight laryngoscope blade 6 Laryngoscope handle and battery holder 7 Nasotracheal tube with funnel 8 Cuffed endotracheal tube with straight slip joint 9 Hemostat to maintain cuff inflation 10 Curved endotracheal tube connector 11 Curved endotracheal tube connector with suction tube nipple 12 Universal metal adapter 13 Catheter Y adapter

result Laryngospasm occurs commonly with extubation but is of less significance if the lungs have been oxygenated prior to removal of the tube

CHOICE OF EQUIPMENT

Figure 22 illustrates most of the essential pieces of endotracheal equipment

Laryngoscope

The type of laryngoscope is not important to the beginner. He can select any of a variety, depending upon his or his supervisor's preference. However, to avoid confusion, he should continue to use one type and develop proficiency before employing another. The student should defer purchasing an instrument until he has had the opportunity to try all of them.

Tubes

There are many types of endotracheal tubes. They are made of natural or synthetic rubber, plastic or metal, a coiled wire may be embedded in the wall to prevent collapse, and they may have an inflatable cuff or balloon built into the wall of the tube or separately attached. Natural rubber has the disadvantage of being porous, thereby accumulating secretions, lubricants and bacteria, which are more difficult to remove. Plastic tubes become firm and stiff with age, and thus more traumatic.

Tubes should be chosen for durability, with thin walls and maximum inside diameter to assure minimum resistance to breathing, lack of compressibility and ease of cleansing. All may link except those of metal or those with coiled wire. Noncollapsible tubes are mandatory in certain types of operations, as for thoracic or cervical tumors obstructing the trachea, and in operations conducted in the face-down position when the head must be sharply flexed.

Endotracheal tubes are longer than needed when received from the manufacturer. They should be cut to lengths such that the distal end of the tube lies 2 to 3 cm. below the larynx with the proximal end at the teeth, in the case of nasotracheal tubes, at the external nares. Nasotracheal tubes are longer than the oral by several centimeters. Endobronchial intubation can result from the use of tubes that are too long. The length of the tube should be estimated for each patient by placement alongside the face and neck. A metal adapter should be inserted

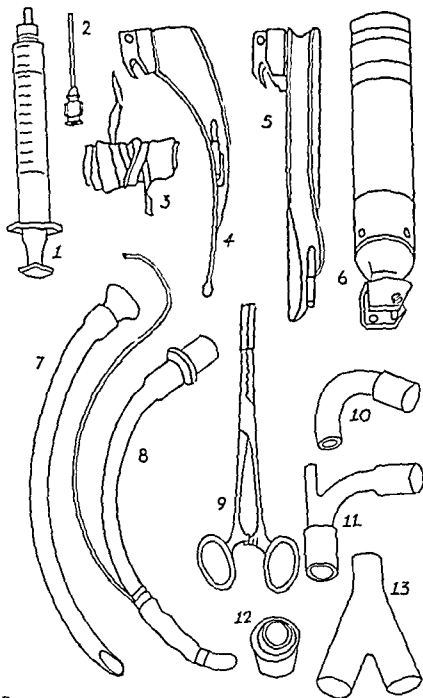


Fig 22 Endotracheal equipment 1 Syringe and (2) blunt tipped needle to inflate cuff on endotracheal tube 3 Bite block 4 Curved laryngoscope blade 5 Straight laryngoscope blade 6 Laryngoscope handle and battery holder 7 Nasotracheal tube with funnel 8 Cuffed endotracheal tube with straight slip joint 9 Hemostat to maintain cuff inflation 10 Curved endotracheal tube connector 11 Curved endotracheal tube connector with suction tube nipple 12 Universal metal adapter 13 Catheter Y adapter

into the end of the tube. This should fit snugly, preferably distending the tube, otherwise it may slip out at an inappropriate time. Connections between tube and anesthetic machine should be large in diameter and with smooth curves to minimize turbulence in air flow, and to decrease resistance.

Stylets should be made of malleable yet firm metal or plastic material. When placed they should not protrude from the tracheal end of the tube. They must be lubricated before insertion into the tube, lest their withdrawal be difficult.

After an endotracheal tube has been inserted, a bite block of rolled gauze should be placed between the teeth. An oropharyngeal airway serves the same purpose. Either will prevent biting on the tube and occlusion of it. The tube should then be fastened to the bite block or airway with adhesive tape and to the cheeks in turn, or with sewing tape tied around the neck. Rarely it may be necessary to suture the tube into place, as in some maxillofacial operations.

Cuffed Tubes

The inflatable balloon or cuff offers the advantage of a completely closed system, thereby making control of respiration more efficient. It prevents passage of foreign material from the pharynx into the lungs. Cuffed tubes may be of the high or low pressure type, so designated according to the pressure needed to inflate the cuff. The latter type is preferable as it lessens pressure and trauma to the tracheal mucous membrane. The cuff should be carefully inflated by a syringe with a measured amount of air, the extent of inflation can be tested by simultaneous compression of the breathing bag and listening for escape of air at the mouth. Pharyngeal packs are not recommended for routine use since they may cause pharyngeal irritation and postoperative sore throat.

Lubricants

In our opinion, uncuffed endotracheal tubes inserted under direct vision through the mouth do not require lubrication. Cuffed tubes should be lubricated to facilitate passage through the glottis. A water-soluble lubricant without local anesthetic is generally adequate but there are some who believe that a lubricant containing a local anesthetic provides worth-while topical anesthesia. Nasotracheal tubes require more than the usual lubrication. Suction catheters should likewise be lubricated for easy passage through endotracheal tubes.

APPRAISAL

The anesthetist should realize that intubation of the trachea is a technique of great value but rarely is it an absolute necessity. Anesthetists have for years administered anesthetics to the satisfaction of all concerned for chest operations, to patients in the prone position and for oropharyngeal surgery without benefit of endotracheal tubes. It is wiser to forego a tube when several attempts at intubation have failed or to ask for help from a more experienced person rather than to persist stubbornly in the face of repeated trauma and failure.

REFERENCES

- Bannister, F. B. and MacBeth, R. G. Direct laryngoscopy and tracheal intubation. *Lancet* 2: 651, 1944.
- Gillespie, N. A. Endotracheal Anaesthesia. University of Wisconsin Press, Madison, Wis., 1950.
- Orkin, L. R., Siegel, M., and Rovenstone, E. A. Resistance to breathing by apparatus used in anesthesia. *Current Researches in Anesth. & Analg.* 33: 217, 1954.
- Pressman, J. J., and Kelemen, G. Physiology of the larynx. *Physiol. Rev.* 35: 506, 1955.
- Proctor, D. F. Anesthesiology and Otolaryngology. Williams and Wilkins Co., Baltimore, 1957.

Intravenous Barbiturate Anesthesia

INDUCTION of anesthesia with the intravenous administration of a rapidly acting barbiturate is pleasant. A patient who has been anesthetized by this method usually prefers it to all others. He relates his experience to friends, who in turn request this technique. Surgeons often promise patients intravenous anesthesia; an enormous demand for the method is thus created. Since the technique is simple, it also appeals to individuals not qualified by experience or training to use it safely. This chapter will outline the principles involved in the safe use of intravenous barbiturates. The two drugs most commonly used are thiopental (Pentothal) and thiamylal (Surital). They are quite similar in their actions and will be treated together. The complications associated with their administration will be emphasized as well.

THEORETIC CONSIDERATIONS

Controllability

Barbiturate anesthesia differs from that produced by inhalational agents in that once the barbiturate has been injected there is little the anesthetist can do to facilitate its removal. With cyclopropane, ether, or other inhalation agents, the blood content can be altered at will by varying the concentration of agent in the respired air. In overdose from inhalant agents, the concentration can be rapidly diminished by ventilation of the lungs with oxygen or air. Controllability of barbiturates is increased by the use of dilute solutions varying from 0.2 per cent to 5.0 per cent. The more dilute solutions are given by continuous infusion while the larger concentrations are injected intermittently.

Theoretically, the lower the concentration, the greater the controllability of the level of anesthesia. Practically, however, since dilute solutions are given by continuous intravenous infusion, there is some hazard in that frequent adjustment of the rate of flow distracts the anesthetist's attention from the patient. Occasionally overdose results because, as flow continues, the anesthetist's attention may be distracted.

Fate in the Body

Whereas all inhalational anesthetics, except trichlorethylene, are inert substances and are eliminated from the body unchanged, the intravenous barbiturate anesthetics must be metabolized and the products of metabolism excreted. The term "ultra short-acting" is still applied to this group of barbiturates. This term is not correct. Apparent brevity of action is related to redistribution of these drugs from the blood stream. They are removed from the circulation promptly and diffuse rapidly into all tissues. Following a single injection, the abundant cerebral blood flow causes the barbiturate content of the brain to rise rapidly. Unconsciousness quickly occurs. As the arterial content decreases with recirculation and as equilibration with other tissues begins, barbiturate is removed from the brain and the concentration in cerebral venous blood may rise above that in the arterial (Fig. 23).

Equilibration with skeletal muscle is attained in about 15 minutes. Fat has a relatively poor blood supply and takes up barbiturate slowly. The maximum deposition in fat usually occurs after 90 to 120 minutes, but uptake may continue for as long as four hours.

After a moderate dose of thiopental (200 to 400 mg) the plasma concentration decreases rapidly and the patient recovers quickly. The larger the amount of drug injected, the greater the tissue storage. Very large doses may be sufficient to saturate the fat depots and there may be a considerable delay in the return of consciousness. When the maximum concentration of the drugs in body fat has been attained, small additional doses will suffice to maintain anesthesia. Failure to recognize this will result in overdosage with occasional serious effects.

The early steep fall in plasma barbiturate concentration is indicative of distribution to fat and other non-nervous tissue. This is followed by a more gradual decline reflecting the slow rate of metabolic transformation of the drugs. In man this rate is only 10 to 15 per cent of the plasma concentration per hour. This can be regarded as a slow rate of metabolism.

Thiopental is almost completely metabolized in man, only about

Intravenous Barbiturate Anesthesia

INDUCTION of anesthesia with the intravenous administration of a rapidly acting barbiturate is pleasant. A patient who has been anesthetized by this method usually prefers it to all others. He relates his experience to friends, who in turn request this technique. Surgeons often promise patients intravenous anesthesia; an enormous demand for the method is thus created. Since the technique is simple, it also appeals to individuals not qualified by experience or training to use it safely. *This chapter will outline the principles involved in the safe use of intravenous barbiturates.* The two drugs most commonly used are thiopental (Pentothal) and thiamylal (Surital). They are quite similar in their actions and will be treated together. The complications associated with their administration will be emphasized as well.

THEORETIC CONSIDERATIONS

Controllability

Barbiturate anesthesia differs from that produced by inhalational agents in that once the barbiturate has been injected there is little the anesthetist can do to facilitate its removal. With cyclopropane, ether, or other inhalation agents, the blood content can be altered at will by varying the concentration of agent in the respired air. In overdose from inhalant agents, the concentration can be rapidly diminished by ventilation of the lungs with oxygen or air. Controllability of barbiturates is increased by the use of dilute solutions varying from 0.2 per cent to 5.0 per cent. The more dilute solutions are given by continuous infusion while the larger concentrations are injected intermittently.

surgical stimulation movement or struggling occurs. Both responses are indicative of poor afferent blockage. Minor pharyngeal and laryngeal stimulation often precipitate laryngospasm or coughing. This is so common that it is said that thiopental sensitizes the vagus and leads to laryngospasm. Actually, we believe this is further evidence that the afferent pathways are unblocked.

Because of the failure to block afferent pathways effectively, intravenous barbiturates usually are combined with inhalational anesthetics, such as nitrous oxide or ethylene. Narcotic analgesics are also frequently given to enhance the analgesic effect of nitrous oxide. With this combination of drugs, however, respiratory depression may likewise be increased.

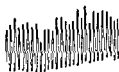
Since intravenous barbiturates do not have the curariform effect of ether and do not effectively block motor impulses, muscular relaxation can be provided only through excessive central depression. This is the reason for the use of relaxant drugs with barbiturate and nitrous oxide anesthesia.

Effect of thiopental upon Respiration

33yr ♂ 68" 142 lb

Control

after 200 mg
thiopental



44	44
46	40

46	47	End exp pCO ₂
37	36	L/min

Respiratory min vol

Fig 24 Spirometric tracings of respiration showing depression caused by thiopental. Compare with respiratory stimulation after ether (Figure 15, page 57).

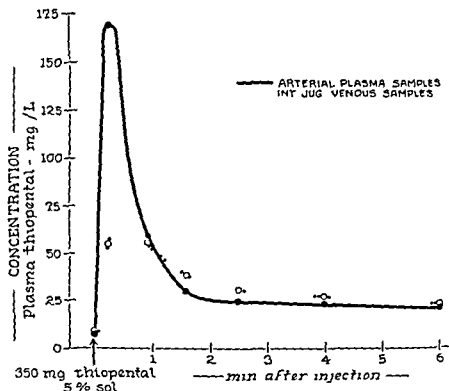


Fig 23 Relation between arterial and cerebral venous blood concentrations of thiopental within first six minutes of an intravenous injection in man.

0.3 per cent of an administered dose being excreted unchanged in the urine. The main site of detoxification of all of the thiobarbiturates is the liver. Narcosis can be expected to last longer in patients with severe liver disease. The role of the kidney is difficult to determine but appears to be less important.

Failure to Block Afferent Impulses

Inhalational anesthetics and intravenous barbiturates differ in their effects upon the central nervous system. Ether, for example, blocks afferent impulses from the periphery, decreases the motor impulses arising in the cortex, and produces a curariform effect at the myoneuronal junction. The degree of effect varies with the depth of anesthesia. Barbiturates block afferent impulses poorly, and have no comparable curariform action.

Recognition of these fundamental differences is essential for the rational use of intravenous barbiturates. Healthy muscular individuals may require excessive doses of barbiturate to produce sufficient depth of anesthesia. Often the degree of anesthesia appears adequate, but with

appear. Because of the alkalinity, the intra arterial injection of these drugs is followed by severe pain and thrombosis.

CLINICAL USE OF INTRAVENOUS BARBITURATES

Selection of Patients

Intravenous barbiturate anesthesia is not suitable for all patients for reasons that are apparent in the foregoing sections. It should be avoided in those with bronchial asthma or other severe allergic phenomena, and in those with advanced cardiovascular disease, or precarious circulatory compensation. Individuals with poor superficial veins are unsuitable. Patients with tumors or other lesions encroaching upon the air passages are unsuitable unless a satisfactory airway is established first, as by the introduction of an endotracheal tube under topical anesthesia. Individuals with acute or chronic respiratory infections and those with productive coughs often react poorly during intravenous barbiturate anesthesia. The method is unsuited to muscular individuals, in other than brief operations, unless supplementation with a potent analgesic and inhalational agent is planned.

Intravenous anesthesia with a barbiturate is not adequate for all types of surgical procedures, even when nitrous oxide or ethylene is added. If the anesthetist chooses this technique or combination, he must be certain that he can provide the proper surgical conditions without giving an overdose. Intravenous anesthesia is a poor choice for long painful operations. It is not suitable where muscle relaxation will be required unless a relaxant drug is given as a supplement.

In recent years a technique of rapidly inducing anesthesia for endotracheal intubation has become popular. With this method 300 to 500 mg. of barbiturate is administered simultaneously with, or followed by, a paralyzing dose of a relaxant drug. This technique is hazardous as is any technique wherein a high concentration of anesthetic is reached within the patient in a short time. If it is used in "poor risk" patients, those with cardiovascular disease, or individuals with high acute intestinal obstruction, the mortality and morbidity may be high. We have seen death, cerebral vascular accidents and myocardial infarction from hypotension following this procedure.

Preparation

Patients are prepared in the manner outlined in Chapters 1 and 5. If narcotic analgesics are administered with preanesthetic medication

Absence of a Respiratory Irritant Effect

The volatile liquid inhalational anesthetics assist in the maintenance of pulmonary ventilation through an irritant effect on the respiratory tract. The intravenous barbiturates lack this attribute, and they do not arouse other stimulant respiratory reflexes, so that respiratory depression may be a prominent feature of anesthesia with these agents (Fig. 24). This is a further reason for giving nitrous oxide and oxygen with barbiturates since a concentration of oxygen above the atmospheric can be maintained.

Parasympathomimetic Action

Under certain circumstances, intravenous barbiturates appear to cause parasympathetic stimulation. This is not common except in those individuals subject to allergic phenomena such as bronchial asthma, urticaria or hay fever. Under these circumstances sneezing, coughing, chest wall spasm, breath-holding, bronchospasm or laryngospasm may follow the intravenous administration of a barbiturate. There are some who consider laryngospasm a parasympathomimetic effect of the barbiturates. We believe, however, that it is more likely the result of the failure of these agents to block afferent impulses originating in the pharynx.

Hypotensive Effect

Hypotension commonly follows the intravenous injection of barbiturates used for anesthesia, the degree depending upon the physical condition of the patient, the amount of the barbiturate used, and the rate of injection. The mechanism of the hypotension has not been clearly defined but probably includes central vasomotor depression and sympathetic ganglionic blockade, as well as direct myocardial depression. While there is frequent mention of a myocardial depressant action of thiopental, recent investigations in heart-lung preparations at least suggest this to be no more pronounced than with other general anesthetics.

Alkalinity of Solutions

Solutions of thiopental and thiamylal are highly alkaline, the pH of a 2.5 per cent or 5.0 per cent solution in distilled water is 10.6. As a result, the intravenous injection of barbiturates may be followed by thrombophlebitis and tissue necrosis in the advent of extravasation. The more dilute the solution, the less likely are these complications to

the first ten to fifteen minutes to assure denitrogenation of the lungs, adequate oxygenation, and initial uptake of anesthetic by the tissues. The flow may be reduced to 2 to 3 l/min after this. If the nitrous oxide or ethylene-oxygen mixture is breathed for ten minutes prior to operation, the total dose of barbiturate required will be less.

Maintenance

The total amount of barbiturate required varies from patient to patient. It depends upon the physical condition of the patient, the preanesthetic medication and the character and duration of the operation. The beginner has difficulty in recognizing the signs of barbiturate anesthesia. Barbiturate is needed with the surgical incision or any other manipulation that may be associated with pain. Quickening of respiration, a rising pulse rate or blood pressure, swallowing and movement of the patient, all suggest the need for more anesthetic. Generally not more than 50 to 100 mg is injected at a time.

Persistent tachycardia, tachypnea and hypertension, alone or in combination, suggest inadequate depth of anesthesia. These may be a manifestation of pain perception. Under such conditions the anesthetist may elect to change to more potent anesthetic agents or he may inject small doses of analgesics intravenously (morphine 2 mg, meperidine 10 mg). Assisted or controlled respiration is necessary if the patient is given relaxant drugs. The indications for additional increments of barbiturate are more difficult to determine under these circumstances. Some anesthetists mix the barbiturate and relaxant drug in the same syringe. This limits the choice of relaxant drug since all are not compatible with barbiturates. Not infrequently, one has the unpleasant experience of having given either too much barbiturate or too much relaxant, usually the latter. In our opinion, use of the agents separately is a better technique.

In some clinics a limit of 1 gm of barbiturate per anesthetic has been established. While this is generally a good rule, it need not be an unalterable one. It becomes a meaningless safety precaution if large amounts of narcotic analgesics are injected in order to limit the total dose of barbiturate.

COMPLICATIONS

Extravascular Injection

In the conscious patient this complication is indicated by pain at the site of injection but in the anesthetized individual it can be detected

the anesthetist can anticipate a greater degree of respiratory depression from the intravenous barbiturate

Anesthesia should not be induced with an intravenous barbiturate unless an anesthetic machine is at hand, with oxygen ready for administration, and the usual assortment of airways available. While it is not necessary to have endotracheal equipment on the machine, it should be available in the immediate vicinity.

Patients who have nasal, pharyngeal or bronchial secretions should be asked to clear these passages before induction. This maneuver will minimize laryngospasm and coughing, so often initiated by secretions irritating the vocal cords after the patient has been anesthetized.

We prefer to inject the barbiturate through the tubing of an intravenous infusion. Some patients have marked venous constriction upon arrival in the operating room. Under these circumstances the anesthetist should not persist in attempts at venipuncture. There is no excuse for exposing a patient to the painful experience of multiple needle punctures in an effort to provide a pleasant induction of anesthesia. It is more sensible to abandon the intravenous route (temporarily at least) and to use an inhalational technique for induction.

Induction of Anesthesia

A test dose of not more than 50 mg. should be given at first. Pain at the site of injection suggests extravasation of the drug. Ordinarily drowsiness will occur with the initial injection. If not, the tourniquet may not have been removed and the drug cannot have reached the general circulation. Absence of drowsiness usually suggests that the patient may require considerable quantities of barbiturate. If loss of consciousness follows the initial dose, the amount of drug subsequently needed for the surgical procedure may be relatively small.

When the barbiturate is to be followed by inhalation anesthesia, enough should be given to permit placement of the mask without the patient being aware of it. If too much barbiturate is given the resulting respiratory depression will slow the induction of inhalation anesthesia.

Intravenous barbiturate is generally administered in combination with nitrous oxide or ethylene for all but brief surgical procedures (closed reduction of fracture or dislocation, incision and drainage of abscess). The combination reduces the amount of barbiturate required and provides a more even level of anesthesia. Either inhalational agent can be used with equal parts of oxygen or up to three parts to one part of oxygen. The patient should breathe at least a 5 l./min. total flow for

Apnea

Cessation of respiration is usually due to an overdose of barbiturate. It is more common if potent analgesics have been administered for preanesthetic medication. Treatment consists of artificial ventilation with bag and mask.

Arterial Hypotension

Deaths have occurred from lowered arterial pressure, usually in patients improperly selected for the technique, or if overdoses have been given. The arterial blood pressure should be carefully observed when barbiturates are administered. Rapid injection or large amounts of drug should be avoided.

Vomiting

This is uncommon during intravenous barbiturate anesthesia unless preceded by coughing, laryngospasm or placement of an airway. Fasting before barbiturate anesthesia is just as important as before inhalation anesthesia.

POSTANESTHETIC COURSE

As a rule recovery from barbiturate anesthesia is gradual and uneventful. The time of awakening is dependent upon total dose, muscular development, physical condition, and the lipid stores. Patients often appear to awaken quickly only to return to an anesthetized state when undisturbed. The possibility of this should not be overlooked. The same postanesthetic precautions must be taken for patients who have been anesthetized with barbiturates as for those anesthetized with inhalational anesthetics.

Patients who have had barbiturates may occasionally show muscular fasciculation, rigidity, stertorous respiration, and slight cyanosis as they recover from anesthesia. This syndrome is poorly understood but appears to be related to temporary derangement of body temperature control. It is more commonly seen when the environmental temperature is low or when the patient has been left uncovered. Treatment consists of the application of warm blankets and inhalation of oxygen.

APPRAISAL

From the foregoing it should be apparent that although the administration of intravenous barbiturates is quick and easy so far as the patient is concerned, it may be a difficult anesthetic technique. The

only by careful observation. Dislodgment is obvious. If extravasation has occurred, treatment consists of injection of 1 per cent procaine into the involved area to lessen the alkalinity, to dilute the barbiturate solution, and to prevent pain and vasospasm. This complication is less common when the injection has been given through the tubing of an intravenous infusion, probably because of dilution.

Intra-arterial Injection

This is a catastrophe. It occurs when a needle is wrongly placed and is detected by severe pain extending into the hand on injection. The barbiturate usually fails to produce sleep but it may if the needle is partly in an artery and partly in a vein. Treatment is urgent. One per cent procaine (10 cc) should be injected through the same needle or through another intra-arterial needle to neutralize and dilute the barbiturate solution. Heparinization through the same needle has also been advised. A stellate block should be performed to promote vasodilatation. If thrombosis of the arteries occurs, as it can after this complication, amputation may ultimately be necessary.

Thrombophlebitis

Thrombophlebitis is usually due to the effect of the alkaline solution causing irritation and thrombosis. It is less likely to occur with 2.5 per cent or more dilute solutions and when the injection is made through the tubing of an intravenous infusion. If the tourniquet has not been removed from the arm before the injection of the barbiturate, the solution will remain in contact with the vessel wall and may cause thrombophlebitis. This is more common when the injection has been given into a leg vein, even with an infusion running. Therefore, the veins of the lower extremity should be avoided.

Coughing and Laryngospasm

Together these are the most common complications of intravenous anesthesia. Often the explanation is poor choice of technique for the patient. One who has witnessed the response of a patient with asthma or chronic bronchitis with coughing, development of chest wall and laryngeal spasm and cyanosis is not apt to forget the experience. Similarly, patients with respiratory tract secretions may behave like this. Blood within the pharynx, stimulation of the air passages with an airway, movement of the head or neck, and painful peripheral stimuli also may be precipitating events.

Spinal Anesthesia

SPINAL anesthesia has been a controversial technique ever since its introduction in 1899. The method consists of the injection of a local anesthetic into the spinal subarachnoid space with resultant anesthetization of spinal ganglia and nerve roots. Many theoretical and practical advantages recommend it. The simplicity of administration long ago made it popular with surgeons who gave the anesthetic themselves. Perhaps this deceptive ease of application has been responsible for many of the complications now feared, in that proper precautions were not taken. Spinal anesthesia is excellent for the performance of intra-abdominal operations. The combination of muscle relaxation, contracted bowel, quiet breathing, and lack of venous congestion is hard to equal with any other method. Even though supplemental general anesthesia is used, the patients are more alert and have less "hangover" postoperatively. The renal circulation is spared more than with general anesthesia. All the desirable attributes of local anesthesia (Chapter 16) are inherent in this technique without the consequence of systemic reaction to the local anesthetic agent.

But there are also certain faults to find. Many patients do not like to be awake during an operation. Another great deterrent in the minds of physician and patient alike is the fear of permanent neurologic damage. The possibility of headache and paralysis contributes to lack of patient acceptance.

After extensive study of spinal anesthesia, we have drawn the conclusion that the complications are no greater in severity than those met with other methods. The kernel of success lies in meticulous preparation of equipment and drugs, and infinite care in technique.

Early difficulties with spinal anesthesia were encountered because

anesthetist must be aware of the problems related here, how best to prevent them and how to treat them should they occur. More important is the knowledge of when to avoid the method even though the patient requests it. The anesthetist should have the skill to use inhalational techniques in a manner that can be equally pleasing to the patient.

REFERENCE

Dundee, J. W. *Thiopentone and Other Thiobarbiturates*. E and S Livingstone, Ltd., Edinburgh and London 1956.

- 3 The anticipated position of the patient during operation
- 4 The patient's preoperative intra abdominal pressure

One can combine the following measures to influence duration, intensity and level of block

Duration of Action of Local Anesthetics

The average duration of anesthesia with the commonly used drugs is 60 minutes for procaine (Novocain), 120 minutes for tetracaine (Pontocaine), and 180 minutes for dibucaine (Nupercaine)

Dosage

The average dose of agent for individuals of varying heights and the desired levels of sensory anesthesia are presented in Table 6 Sacral

Table 6

BODY LENGTH (Inches)	ANESTHETIC LEVEL	PROCAINE* (mg)	TETRACAINE† (mg)	DIBUCAINE‡ (ml)
60	Sacral	50	4	4
	Groin	75	8	8
	Xiphoid	100	12	10
	Nipple	125	14	12
66	Sacral	50	6	6
	Groin	100	10	10
	Xiphoid	150	14	14
	Nipple	175	16	16
72	Sacral	75	8	8
	Groin	125	12	12
	Xiphoid	175	16	16
	Nipple	200	20	20

* Procaine 5% in cerebrospinal fluid

† Tetracaine 1% sol with 10% dextrose equal volumes

‡ Dibucaine 1:1500 sol (1 ml = 0.66 mg)

levels permit the performance of operations about the perineum, levels at the groin allow for operation on the legs and thighs, xiphoid levels permit lower abdominal procedures, and sensory levels at the nipple are for upper abdominal operations. It must be remembered that the upper level of anesthesia is only a sensory level and that motor nerves to muscle permitting muscular relaxation are blocked at least several dermatomes below the sensory nerves. This is because of the differential blocking action of local anesthetics on nerve fibers of varying size (See Chapter 16)

the physiologic alterations secondary to interruption of nerve conduction in the subarachnoid space were poorly understood. This has now been largely overcome. Arterial hypotension, for example, has been shown to follow failure of peripheral vasoconstriction or decreased output of the heart as the result of inadequate venous return. A third possibility is denervation of the adrenal medulla and lack of synaptic transmission, both followed by a deficiency in circulating pressor substances. It is conceivable that all three may be operative at the same time but the cause of hypotension lies chiefly in interruption of efferent impulses in the sympathetic nervous system with resultant arteriolar and venous dilatation.

Respiratory inadequacy may be associated with spinal anesthesia just as with other methods. The cause is a high level of anesthesia leading to block of the intercostal nerves, or, if the level is exceptionally high, to paralysis of the phrenic nerve roots. These possibilities must be recognized and treated just as if the patient were curarized.

PREANESTHETIC APPROACH

During the preanesthetic visit a patient should be told that he will be given spinal anesthesia. Whenever fears are expressed and are irreconcilable another method of anesthesia should be substituted. The frightened patient will implicate spinal anesthesia in any complication that occurs. A careful history should be taken to elicit symptoms of neurologic disease, even at the expense of suggesting the development of these. Frequent headache, backache, past or present neurologic disease of congenital, infectious or traumatic origin should preclude selection of spinal anesthesia. One must not risk exacerbation of pre-existing disease. At this time the patient's back should be examined to detect structural or other conditions that might make lumbar puncture difficult or hazardous. Superficial infections, ankylosis, congenital abnormalities and severe curvatures of the spine are some of the things to look for.

AGENTS AND TECHNIQUES

The composition of the solution to be injected into the subarachnoid space and the technique used for injection are determined by the following factors

- 1 The duration, intensity and level of block desired
- 2 The length of the vertebral column of the patient

- 3 The anticipated position of the patient during operation
- 4 The patient's preoperative intra-abdominal pressure

One can combine the following measures to influence duration, intensity and level of block

Duration of Action of Local Anesthetics

The average duration of anesthesia with the commonly used drugs is 60 minutes for procaine (Novocain), 120 minutes for tetracaine (Pontocaine), and 180 minutes for dibucaine (Nupercaine)

Dosage

The average dose of agent for individuals of varying heights and the desired levels of sensory anesthesia are presented in Table 6. Sacral

Table 6

BODY LENGTH (Inches)	ANESTHETIC LEVEL	PROCAINE* (mg)	TETRACAINE† (mg)	DIBUCAINE‡ (ml)
60	Sacral	50	4	4
	Groin	75	8	8
	Xiphoid	100	12	10
	Nipple	125	14	12
66	Sacral	50	6	6
	Groin	100	10	10
	Xiphoid	150	14	14
	Nipple	175	16	16
72	Sacral	75	8	8
	Groin	125	12	12
	Xiphoid	175	16	16
	Nipple	200	20	20

* Procaine 5% in cerebrospinal fluid

† Tetracaine 1% sol with 10% dextrose equal volumes

‡ Dibucaine 1:1500 sol (1 ml = 0.66 mg)

levels permit the performance of operations about the perineum, levels at the groin allow for operation on the legs and thighs, xiphoid levels permit lower abdominal procedures, and sensory levels at the nipple are for upper abdominal operations. It must be remembered that the upper level of anesthesia is only a sensory level and that motor nerves to muscle permitting muscular relaxation are blocked at least several dermatomes below the sensory nerves. This is because of the differential blocking action of local anesthetics on nerve fibers of varying size (See Chapter 16).

Addition of Vasopressor Drug

A vasopressor drug as part of the solution injected into the subarachnoid space will increase the duration of anesthesia. We prefer 0.3 to 0.5 mg of epinephrine for this purpose, and hope for a 50 to 100 per cent increase in duration of anesthesia. Despite the theoretic objection that nerve tissue damage might follow the diminished blood supply secondary to vasoconstriction, this technique has proven safe in our hands.

Concentration of Local Anesthetic

If sensory block alone is sought, a lesser concentration of anesthetic is needed than if conduction in the larger, more heavily myelinated motor fibers is to be interrupted. If muscular relaxation, therefore, is not essential a weaker solution of anesthetic can be used.

Specific Gravity of Solution

The specific gravity of cerebrospinal fluid is between 1.004 and 1.008. By dissolving the local anesthetic in distilled water or 0.5 per cent saline a solution with a specific gravity less than that of cerebrospinal fluid (hypobaric) is achieved. By using cerebrospinal fluid or 10 per cent dextrose as the diluent a hyperbaric solution results. Differences in specific gravity, when correlated with the patient's position during and after injection, can be used to assist in the spread of the anesthetic in the subarachnoid space. If, for example, a hyperbaric solution is selected and the patient sits up during and after injection, a low level of block can be expected. Injection of a hypobaric solution under the same circumstances would result in a high level of anesthesia. Because of the curves in the spinal column (Fig. 24) a hyperbaric solution will tend to reach the third to sixth thoracic segments if the patient is placed on his back. The common mixture of equal volumes of 1 per cent tetracaine and 10 per cent dextrose with a specific gravity of 1.023 is most likely to do this, despite attempts to limit the level of block.

The final selection of solution and technique is reached in this way. The taller the patient, and therefore the greater the spinal cord length, the larger the volume and amount of drug needed to accomplish a given purpose. Increased intra-abdominal pressure is an indication for a reduction in volume and amount of the local anesthetic injected. Elevated intra-abdominal pressure causes back pressure in epidural veins which, as they dilate, encroach upon the subarachnoid space and narrow it. The anesthetic solution therefore is injected into a lesser

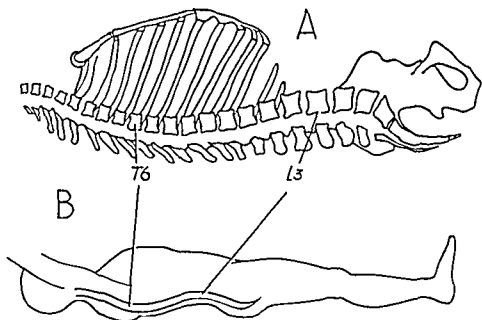


Fig 24 Spinal column curvatures that influence the spread of anesthetic

volume of cerebrospinal fluid, and spreads over a wider area. Full-term pregnancy, ascites, abdominal tumors, intestinal distention and obesity are instances in which abdominal pressure is likely to be above normal.

The position of the patient on the operating table is an additional factor in the choice of anesthetic solution. Hypobaric solutions are of value for operations carried out with a patient in the lateral flexed (kidney) position. Injection can be made with the operative site uppermost. If the Trendelenburg position is to be used, and a low level of anesthesia will suffice, as for vaginal procedures, use of hypobaric solution will minimize the likelihood of an unnecessarily high block. If a cholecystectomy is the procedure, a high, solid block is needed and a hyperbaric solution in our opinion is most reliable for this. These examples illustrate the effect of patient position on the technique elected.

Provision for Serial Injection

A plastic catheter or a malleable needle may be left in the subarachnoid space during operation. Additional quantities of anesthetics are injected as needed. Such a technique provides almost unlimited duration of anesthesia. It also affords greater controllability of dosage,

particularly in the ill patient in whom selection of a proper dose for single injection may be difficult. We also believe that the injection of anesthesia in small increments lessens the rapidity of onset and degree of hypotension. This is a more difficult technique perhaps with a greater incidence of postanesthetic neurologic sequelae, but there are certain advantages to its use.

SPINAL ANESTHETIC EQUIPMENT

Proper preparation and sterilization of equipment are most important in the prevention of neurologic sequelae. Everything to be injected into the subarachnoid space can be packaged in one tray. If a choice of anesthetics is desired, the drugs can be packed and autoclaved separately and added to the trays as needed. The tray (Fig 25) should contain

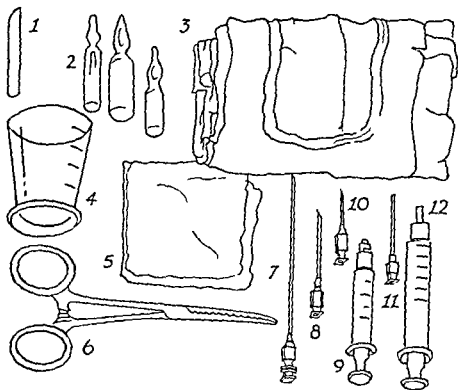


Fig 25 Contents of a spinal tray 1 Ampule file 2 Ampules containing pressor drug local anesthetic and spinal anesthetic. 3 Drape for back. 4 Medicine glass 5 Gauze sponges 6 Hemostat to prepare back. 7 Lumbar puncture needle 8 Intramuscular needle 9 Syringe for local anesthesia of the back. 10 Hypodermic needle 11 Mixing needle for spinal anesthetics 12 Syringe for spinal anesthetics. Other material such as gloves and sterilization indicators may be incorporated in the tray before autoclaving.

- 1 Surgical gloves and towel
- 2 Spinal drape (circumcision type for the patient's back)
- 3 Medicine glass, to hold ampules
- 4 One 5 ml plain tip syringe for the solution to be placed in the subarachnoid space
- 5 One blunt tip #18 gauge mixing needle for the spinal anesthetic
- 6 One 2 ml Luer-Lok syringe for procaine infiltration of the back
- 7 One #25 gauge hypodermic needle for infiltration of the skin
- 8 One #22 gauge lumbar puncture needle, 3 1/2 inches, with stylet
- 9 One #22 gauge needle, 2 inches, for intramuscular injection of local anesthetic and pressor drug
- 10 One sponge forceps and sponge for preparing the skin of the back
- 11 One ampule file
- 12 An ampule of procaine for local anesthesia and an ampule of a pressor drug

The trays should be used only for the performance of spinal anesthesia. A trustworthy attendant should be assigned the task of cleaning and rinsing needles and syringes so that contaminating substances, particularly blood, and the cleansing solutions are removed entirely before autoclaving. Local anesthetics should be purchased from reputable pharmaceutical firms. When the trays have been assembled and wrapped they should be autoclaved for 20 minutes at a temperature of 260° F and 27 lbs pressure. Trays should be allowed to dry in the autoclave before removal and should be marked with the date of sterilization. Trays are redone if not used within two weeks.

If ampules of substances to be introduced into the subarachnoid space are wrapped separately, they are autoclaved at the same temperature and pressure but for only ten minutes. This includes epinephrine, the potency of which does not appear to be significantly reduced by such treatment. If an autoclaved ampule is not used within two weeks, it is discarded. Drugs are not re-autoclaved. Sterilization of ampules by immersion in antiseptics should never be practiced, for the antiseptic may contaminate the local anesthetic or pressor drug if the ampule is cracked. Detection of contamination is not always possible and neurologic damage may follow the use of such a solution.

ANCILLARY PREPARATIONS

These are the same as for any anesthetic. It is especially important that an anesthetic machine be in readiness for the treatment of respira-

particularly in the ill patient in whom selection of a proper dose for single injection may be difficult. We also believe that the injection of anesthesia in small increments lessens the rapidity of onset and degree of hypotension. This is a more difficult technique perhaps with a greater incidence of postanesthetic neurologic sequelae, but there are certain advantages to its use.

SPINAL ANESTHETIC EQUIPMENT

Proper preparation and sterilization of equipment are most important in the prevention of neurologic sequelae. Everything to be injected into the subarachnoid space can be packaged in one tray. If a choice of anesthetics is desired, the drugs can be packed and autoclaved separately and added to the trays as needed. The tray (Fig. 25) should contain

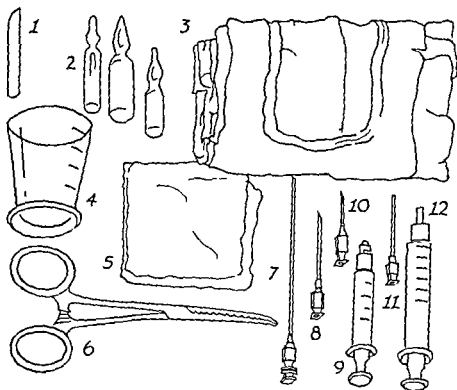


Fig. 25 Contents of a spinal tray 1 Ampule file 2 Ampules containing pressor drug local anesthetic and spinal anesthetic 3 Drape for back 4 Medicine glass 5 Gauze sponges 6 Hemostat to prepare back 7 Lumbar puncture needle 8 Intramuscular needle 9 Syringe for local anesthesia of the back 10 Hypodermic needle 11 Mixing needle for spinal anesthetics 12 Syringe for spinal anesthetics. Other material such as gloves and sterilization indicators may be incorporated in the tray before autoclaving.

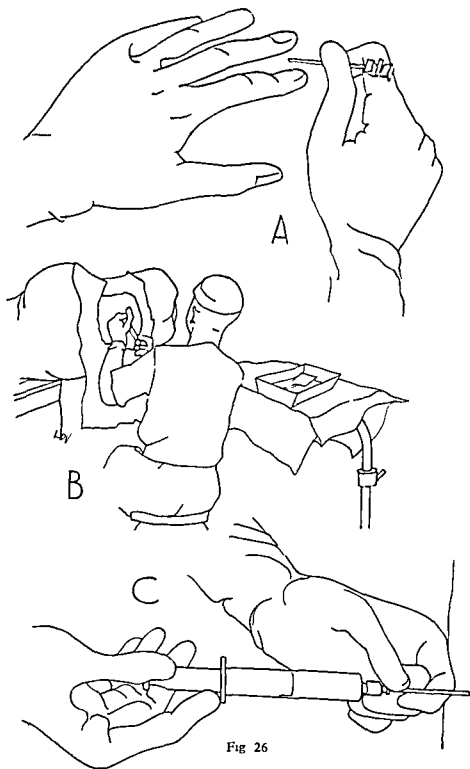


Fig 26

tory emergencies. An intravenous infusion may be started beforehand in patients with precarious circulation or instituted later to combat arterial hypotension and to provide an avenue for supplemental anesthesia when necessary.

TECHNIQUE OF LUMBAR PUNCTURE AND DRUG INJECTION

The lateral decubitus is the best routine position for performance of lumbar puncture. The side to be operated upon should be lowermost if hyperbaric solutions of anesthetics are to be given and uppermost if hypobaric techniques are chosen. If lumbar puncture promises to be difficult, the sitting position may better accentuate the bony landmarks. In any case flexion of the lumbar spine is important to open the vertebral interlaminar spaces and to separate the vertebral spines. An assistant should maintain gentle steady flexion of the back, support the patient and prevent indecent exposure of the patient. Before donning his gloves the anesthetist should palpate the lumbar spine, and select and mark a promising vertebral interspace. An imaginary line between the iliac crests should intersect the spine at the third or fourth lumbar interspinal space, both of which are well below the expected level of termination of the spinal cord at lumbar space two.

The spinal tray is opened on a small table. Gloves are removed from the tray and donned with care to avoid breaks in technique. The anesthetist's hands should have been washed and dried but need not be surgically clean. The equipment on the tray is approached with a "no touch" technique, that is the barrel of the syringe used for the spinal anesthetic and the needle to be introduced into the subarachnoid space should not be handled. Ampules are carefully inspected for imperfections and the solutions for clarity. Suspicious ampules should be discarded. Ampules are opened with a file to avoid splintering of glass and requisite quantities of anesthetic are drawn up and mixed in the designated syringes. The lumbar puncture needle must be reexamined for sharpness and cleanliness. A needle of small diameter should be selected since lumbar puncture headache results from leakage of cerebrospinal fluid through the dural puncture opening; the larger the needle, the larger the opening and the greater the leakage. We have found a 22 gauge needle satisfactory for routine work and teaching.

Fig 26 Details of spinal anesthesia technique. A Insertion of lumbar puncture needle with right hand. The left hand fixes the skin over the chosen lumbar intervertebral space. B Proper position for lumbar puncture. Anesthetist is sitting with his eyes at the level of the lumbar spine. Spinal tray on separate table to the right. C Proper position of hands to connect syringe to lumbar puncture needle and to aspirate and inject fluid.

If bone is met the needle should be withdrawn to the subcutaneous tissues and redirected. The commonest causes of failure are improper position of the patient, selection of the wrong interspace (usually one over the sacrum) or failure to advance the needle in the midline.

When the dura has been penetrated, withdrawal of the stylet should be followed by a free flow of clear cerebrospinal fluid. Rotation of the needle will place the needle bevel well within the subarachnoid space and disengage soft tissues at the tip. Bloody fluid or lack of free flow contraindicates injection of the spinal anesthetic. Blood-tinged fluid which clears is not a contraindication to the subsequent injection of anesthetic. One should never inject an anesthetic when paresthesias are present since the needle tip may lie within or against a nerve root. Permanent neurologic damage may follow an intraneural injection.

Having satisfied the requirements of an atraumatic puncture one is ready to inject the anesthetic. While the needle is supported with one hand against the back, the other hand firmly attaches the syringe tip to the needle hub (Figure 26). If cerebrospinal fluid can be easily withdrawn into the syringe, the anesthetic is injected at an even rate of approximately 1 ml in 5 seconds. Syringe and needle are withdrawn slowly as a unit after a final demonstration of free flow of cerebrospinal fluid into the syringe. Barbotage should be avoided.

Following Injection

The period immediately following injection is a crucial time. The patient is positioned for operation. Straining, breath-holding or movement on the part of the patient may raise the level of anesthesia to unexpected heights. Blood pressure may fall. For these reasons the anesthetist must immediately devote his full attention to the patient, testing the level of anesthesia with pinprick and alternately measuring the blood pressure. All other matters are of secondary importance. The assistant can arrange the drapes, ether screen, operating room lights and arm rest. The anesthetist should record his findings and can tilt the operating table in one direction or another, depending upon the baricity of the anesthetic if the level of anesthesia is not satisfactory. Levels must be checked if possible for at least the first half hour after injection. Motor power is assayed by asking the patient to move his toes, or to tense the rectus muscles by raising the head briefly. The anesthetist's fingers placed over the intercostal spaces as the patient breathes deeply will detect intercostal paralysis.

The onset of spinal anesthesia is noted within one to two minutes of injection as a rule. Occasionally patients are seen in whom a delay

Smaller gauge needles cannot be inserted into the tough tissues of the back. When fine needles are used, larger introducing needles are needed and the technique is correspondingly more difficult.

The patient should be forewarned before each maneuver, this leads to greater patient confidence and cooperation. When everything has been prepared on the tray, the skin of the patient's back should be prepared with a colored antiseptic. One must avoid contamination of needles and drugs that are placed into the subarachnoid space. An assistant can pour the antiseptic onto the sponge stick which is held by the anesthetist over a waste bucket, or the anesthetist can dip his sponge into a cup of antiseptic placed well away from the spinal tray. Beginning at the lumbar puncture site and working concentrically, the skin of the back is painted over a wide area. The sponge stick is set aside at the completion of the prep and the back covered with the sterile drape, so placed that the opening overlies the lumbar spine.

The anesthetist should be seated with the patient's spine at eye level (Fig. 26, B). The skin must not be touched until dry to avoid wetting the gloves and contaminating syringes and needle. A small intracutaneous skin wheal of procaine is raised at the selected interspace using the 23 gauge needle and the small Luer-Lok syringe. Infiltration is carried in the midline down to the supraspinous ligament. This is followed by the intramuscular injection of a pressor drug to avoid or minimize the expected fall in blood pressure which follows anesthetization of sympathetic vasoconstrictor fibers in the subarachnoid space. The 22 gauge intramuscular needle and 2 ml. syringe are used for this purpose with injection through the skin wheal into the sacrospinalis muscles. The pressor drug used and the dose chosen are a matter of individual preference and experience (see Chapter 20). Placing the pressor drug intracutaneously or into the ligaments of the spinal column limits the absorption of the pressor drug and hence its effectiveness.

Introduction of the lumbar puncture needle (Fig. 26, 4) must be performed deliberately and carefully to avoid injury to the soft tissues, ligaments and periosteum. Traumatic lumbar punctures are probably responsible for 90 per cent of complaints after spinal anesthesia which include headache, backache and sciatic radiation of pain. With the stylet in place the needle is advanced in the midline through the skin wheal in a direction perpendicular to all planes of the back, or slightly cephalad. The tissues penetrated are the supraspinous and interspinous ligaments, the ligamentum flavum and finally the dura. After experience the beginner learns to distinguish these structures by a sense of touch.

needed. Often it is the fear of being awake or of the anesthetic ending prematurely that makes the patient reject spinal anesthesia. Nausea and vomiting are common. The action of narcotics used for preanesthetic medication, cerebral ischemia, psychic upset, traction on viscera, or the central stimulation of pressor drugs may be responsible for the gastrointestinal symptoms. These can be treated with intravenous sedatives or resort to general anesthesia with nitrous oxide and intravenous thiopental.

Fate of Local Anesthetics Placed within the Subarachnoid Space

After injection of the local anesthetic into the subarachnoid space, the concentration in spinal fluid declines rapidly. Analysis of tissues affected by the anesthetic indicates that the concentration in spinal nerve roots and ganglia is high. Small amounts of drug penetrate the spinal cord but impairment of conduction in the cord does not appear to be an essential part of spinal anesthesia. The local anesthetic can be found in the blood stream as soon as the injection is completed, with venous drainage the major route of departure. Only a small quantity leaves via the lymphatic system.

In the blood stream the drug is distributed to the body tissues, but only kidney and liver are able to concentrate it above the blood level. Hydrolysis, conjugation or detoxification of the local anesthetic may take place in the circulating plasma but does not occur in the cerebrospinal fluid. The urine provides the main pathway of removal from the body. These facts have been established for procaine but may apply to other local anesthetics as well.

DELAYED SEQUELAE OF SPINAL ANESTHESIA

The syndrome of decreased intracranial pressure consisting primarily of headache, but occasionally including difficulties in hearing and vision, is the most common complication of lumbar puncture and spinal anesthesia. The opening in the dura (Fig. 27) made by the lumbar puncture needle may persist for days (Fig. 28) or weeks. Cerebrospinal fluid leaks out through this hole. Formation of cerebrospinal fluid may not keep pace with the loss and spinal fluid pressure falls.

Headache

Evidence is overwhelming that reduction in cerebrospinal fluid pressure is followed by a characteristic headache, appearing with assumption of the erect position, and usually relieved by recumbency.

of 15 to 20 minutes is observed, perhaps because of slower diffusion and penetration of the anesthetic through the nerve roots. Failure of anesthesia to develop is most commonly related to the fact that the tip of the lumbar puncture needle was not properly within the subarachnoid space. Impotent local anesthetics or inadvertent omission of an anesthetic from the injected solution may be implicated. The term "rachis-resistance" has been coined to describe a failure with spinal anesthesia, but, admitting that in some individuals a slow onset of anesthesia occurs, we believe technical errors are responsible in the majority of instances.

A patient under spinal anesthesia needs the same careful observation as one under general anesthesia. Respiratory inadequacy due to a high level of spinal anesthesia may be difficult to detect if only the lower intercostal muscles have been paralyzed. If the slightest suspicion is aroused however, oxygen should be given by mask since prolonged minor degrees of hypoxia may terminate in sudden circulatory failure. Higher levels of intercostal paralysis may be accompanied by a feeling of suffocation and signs of sensory and motor paralysis in the arms. When the phrenic nerve roots in the cervical region have been blocked, accessory muscles of respiration in the neck will be called into action. The patient loses his voice for lack of ability to move air and he may lose consciousness. Total paralysis of respiration is a dire emergency. Artificial respiration with oxygen and mask or endotracheal tube must be carried out until the level of anesthesia has receded. The circulation must be supported simultaneously.

Arterial hypotension may be said to be present during spinal anesthesia when there has been a marked lowering of the preoperative values or if the systolic blood pressure falls below 80 mm Hg. Development of hypotension is more hazardous in the hypertensive patient accustomed to a high level of pressure. Hypotension is most dangerous if coronary or cerebral insufficiency has been noted in the past. The immediate treatment of this complication consists of giving oxygen by mask and the intravenous injection of a pressor drug. Suggested dosages may be found in the chapter on arterial hypotension. Only small amounts should be given intravenously to avoid excessive elevation of pressure. At the same time a larger intramuscular injection should be given for a sustained effect. If frequent injections of pressor drug are required a continuous infusion of phenylephrine (Neo-synephrine) may sustain a more steady level of pressure.

The anesthetist should never leave the head of the table. Vocal encouragement or supplementation with light general anesthesia may be

Presumably the ache is caused by loss of the cushioning effect of the fluid, with downward displacement of the brain and traction or compression on such pain sensitive intracranial structures as nerves and blood vessels

In a series of 9277 spinal anesthetics studied by the authors the overall incidence of headache was 11 per cent. Headache occurred decreasingly after the fifth decade, with the greatest frequency in the third and fourth decades (Table 7). This difference among age groups

Table 7 Relation of the Age of the Patient to Incidence of "Spinal" Headache

AGE (YEARS)	NUMBER OF SPINAL ANESTHETICS	SPINAL HEADACHES Number	Per cent
10-19	537	51	10
20-29	1994	321	16
30-39	1833	261	14
40-49	1759	192	11
50-59	1736	133	8
60-69	1094	45	4
70-79	297	7	2
80-89	27	1	3
	9277	1011	11

may be attributed to an elevation of pain threshold in the aged, perhaps to a progressive decrease in sensory neural elements with increasing age. There may also be a decrease in elasticity of the cerebral vessels with aging.

The incidence of headache is much greater in females (Table 8). In part this is related to inclusion of obstetric cases in the female group for headache followed vaginal delivery with the patient under

Table 8 Relation of the Sex of the Patient to the Incidence of "Spinal" Headache

	NUMBER OF SPINAL ANESTHETICS	SPINAL HEADACHES Number	Per cent
Male	4063	302	7
Female	5214	709	14
	9277	1011	11



Fig 27 Puncture openings in the dura Magnification 50X. Note the difference when the bevel of the needle is at right angles to the fibers of the dura (left) or parallel to the fibers (right)

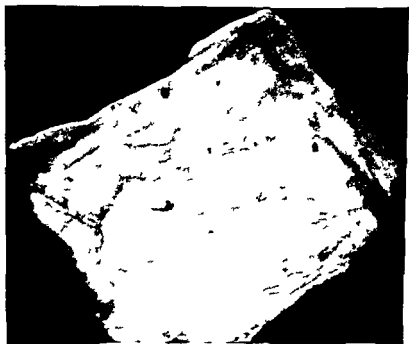


Fig 28 Puncture opening in the dura, two days old Diameter of needle 0.8 mm Magnification 3X. (From Franksson and Gordh *Acta chirurg Scandinav* vol 94, 1946)

negligible complication after spinal anesthesia by the use of a 26 gauge needle for lumbar puncture, and by the oral or parenteral administration of more than usual amounts of fluid. In our series, a 16 gauge needle was employed for the introduction of a ureteral type of catheter to produce continuous spinal anesthesia. Not only were the technical difficulties greater but the incidence and severity of headache and ocular complaints were so prohibitive that we soon restricted this technique to the older age groups, wherein headache was not as much of a factor, or the method was reserved for those situations in which the greater safety and better control with the continuous technique were a major consideration. For routine use and freedom from technical difficulty, a 22 gauge needle proved to be best. The over-all incidence of headache with this needle was nearly 9 per cent, but the headaches were generally mild.

If headaches do occur, treatment can be directed logically at the pain sources within the cranium with pharmacologic agents, or therapy can be directed toward increasing cerebrospinal fluid pressure. Increased bodily hydration or the application of a tight abdominal binder will sometimes relieve the milder headache. A second lumbar puncture with replacement of fluid will alleviate headache immediately. The reasons are not clear, but sometimes relief will be permanent after this maneuver, despite the fact that the dura has been punctured a second time. Neither is the mechanism clear whereby the epidural placement of fluid is said to relieve headache. It may be due to a cushioning effect on the dural column of fluid. There is evidence that some of the epidural fluid traverses the fistula to raise cerebrospinal fluid pressure directly. Another possibility is the production of a reparative reaction in the epidural tissues, enough to close the opening. A word of caution must be added against the practice of injecting solutions indiscriminately into the subarachnoid space to relieve headache. Whenever a foreign substance is added to the cerebrospinal fluid there is the hazard of meningitis or arachnoiditis. We have heard of signs of irritation in the cauda equina after the epidural placement of saline solution. Direct artificial elevation of the cerebrospinal fluid pressure should be reserved for the treatment of incapacitating headaches when nausea, vomiting or dizziness is protracted and when an ocular nerve palsy seems imminent.

Difficulties with Hearing

Changes in hearing occurred in 0.4 per cent of our series of 9277 spinal anesthetics. These consisted of complaints of buzzing, popping, clogging of the ears, humming, roaring or loss of hearing altogether.

spinal anesthesia in 22 per cent of the series, twice the over-all figure. The reasons for this may be extreme changes in intra-abdominal pressure during labor, which could tend to increase leakage and keep the dural fistula open, rapid changes in blood volume following delivery, dehydration during labor and the lesser attention paid to paracenteral fluid replacement after delivery. Yet even if the obstetric cases are removed from consideration, headache still proved more of a problem in women (Table 9). It is difficult to relate this to a con-

Table 9 Incidence of "Spinal" Headache According to Sex if Patients Receiving Spinal Anesthesia for Vaginal Delivery Are Excluded

	NUMBER OF SPINAL ANESTHETICS	SPINAL Number	HEADACHES Per cent
Male	4063	302	7
Female	4276	489	12

stitutional variation between men and women in regard to pain perception or susceptibility. The role of the psyche in the development of headache in either sex seems negligible. To prove this point we gave spinal anesthetics to 100 persons anesthetized first with general anesthetics. Although none of these people knew that spinal anesthetics had been administered, the percentage occurrence of typical postural headache was the same as in the series at large.

Table 10 indicates that the incidence of headache diminishes progressively with smaller needle diameters. There was not only a lesser occurrence but a lesser severity and duration. Headache can be made a

Table 10 Relation of Gauge of Needle Used for Lumbar Puncture to Incidence of "Spinal" Headache

NEEDLE GAUGE	NUMBER OF SPINAL ANESTHETICS	SPINAL Number	HEADACHES Per cent
16	839	151	18
19	154	16	10
20	2698	377	14
22	4952	430	9
24	634	37	6
	<u>9277</u>	<u>1011</u>	<u>11</u>

Major Neurologic Sequelae

In 1950 Foster Kennedy wrote, "From a neurological point of view we give the opinion that spinal anesthesia should be rigidly reserved for those patients unable to accept a local or general anesthetic. Paralysis below the waist is too large a price for a patient to pay in order that the surgeon should have a fine, relaxed field." The sequela most feared is chronic progressive adhesive arachnoiditis, which we believe is a non-specific pathologic response to an intrathecal irritant. The ultimate effects of chronic progressive adhesive arachnoiditis are related to a reduction of blood supply to the spinal cord. Transverse myelitis has also been described. When this occurs, it would seem to be related more to destruction of neural tissue by the injected substance or to an intraspinal or intraneural injection than to fibrosis resulting from an irritant. The end results of either of these two processes can be paralysis of the lower limbs, intestines and bladder or involvement of spinal cord and roots at higher levels. Sensory changes alone may occur, they obviously are less disturbing and incapacitating.

In our large series of spinal anesthetics, accurate information about 86 per cent of the patients was obtained for at least six months after anesthesia. In some the follow-up period exceeded seven years. There were no instances of adhesive arachnoiditis, cauda equina syndrome or transverse myelitis.

Minor Sensory Neurologic Sequelae

Residual signs or symptoms of numbness following spinal anesthesia occurred in 0.8 per cent of the series. In the majority of patients this was restricted to the lumbar and sacral areas of the body. None of the cases had other neurologic signs, and most of the complaints disappeared within six months. Subjective complaints were those of numbness, numbness and tingling, heaviness or burning. Sensory defects were not always found on examination.

It seems likely that these sequelae are related to the injection of the anesthetic drug into the subarachnoid space. Increased numbers of cells in the cerebrospinal fluid following spinal anesthesia suggest that a transient sterile type of myelitis or meningitis may be produced during spinal anesthesia. Neurologic sequelae have followed the injection of various foreign substances into the subarachnoid space. The delicacy of spinal structures and their peculiar vascularity almost invite trouble if there be trespass. The encouraging aspect of the cases reported here is the minimal nature of the resultant disease. We feel certain that this good fortune is due to the careful application of spinal

With few exceptions these troubles were associated with typical postural headache, hence a decrease in cerebrospinal fluid pressure must be implicated once more. That there is an anatomic communication between the subarachnoid space and the cochlea is well known. Experimental work by Hughson demonstrated that with a decrease in cerebrospinal fluid pressure, there was a fall in intralabyrinthine pressure followed by a functional inability of the ear to transmit high tones. On several occasions we have seen restitution of an audiogram to normal values when cerebrospinal fluid pressures were artificially elevated toward normal in individuals with headache and decreased hearing after spinal anesthesia. One might assume that, in addition to deafness, the other auditory complaints after spinal anesthesia might be secondary to a decreased cerebrospinal fluid pressure.

Ocular Problems

Difficulty with vision also occurred in 0.4 per cent of the series. Double vision, blurring, troubles in focusing and spots before the eyes were the complaints. In all but 8 of the 34 patients the visual trouble arose in association with typical postural headaches. We discovered 3 proved cases of lateral rectus muscle paralysis, and the symptom of prolonged double vision in 3 other persons suggested that paralysis might also have been present. These 6 cases followed the use of a 16 gauge needle for continuous spinal anesthesia given through a catheter. Five of the 6 patients had severe headaches, in 3 localized to the occipital area. One cannot help but relate the palsies and the other ocular complaints to headache and consequently to a decrease in intracranial cerebrospinal fluid pressure. The ocular nerve palsies, in our experience, came on rather suddenly a week or so after operation and persisted from a few weeks to six months. In every case there was complete functional restitution of vision. It must be assumed that an ocular nerve palsy represents an end stage of whatever ocular abnormality is present. Therefore, more benign ocular complaints may represent phases or steps toward the final result without progression. There have been many reported cases of cranial nerve palsy arising after spinal anesthesia, but the abducens nerve usually has been involved. One theory of origin has been that, with brain displacement and traction on supporting structures, a motor nerve particularly one with a long intracranial course, could be paralyzed by pressure against osseous structures, or, in the passage of the nerve through the cavernous sinus excessive venous dilatation might produce the same effect.

REFERENCES

- Bonica J J, Backup P H, Hadfield, D, Crepps, W F, Monk, B F, and Anderson, C. L. Peridural block. Analysis of 3637 cases and a review. *Anesthesiology* 18 Sept, 1957
- Dripps, R. D., and Deming M. van N. An evaluation of certain drugs used to maintain blood pressure during spinal anesthesia. Comparison of ephedrine, paredrine, pitressin-ephedrine and methedrine in 2500 cases. *Surg, Gynec & Obst.*, 83 312, 1946
- Dripps R. D. and Vandam L. D. Hazards of lumbar puncture. *J A M A*, 147 1118, 1951
- Dripps R. D., and Vandam L. D. Long term follow up of patients who received 10 098 spinal anesthetics. I. Failure to discover major neurological sequelae. *J A M A*, 156 1486 1954
- Macintosh R. R. Lumbar Puncture and Spinal Analgesia. E & A Livingstone, Ltd. Edinburgh, 1951
- Vandam, L. D. and Dripps R. D. A long term follow up of 10 098 spinal anesthetics. II. Incidence and analysis of minor sensory neurological defects. *Surgery*, 38 463 1953
- Vandam L. D., and Dripps, R. D. Long term follow up of patients who received 10 098 spinal anesthetics. Syndrome of decreased intracranial pressure (headache and ocular and auditory difficulties). *J A M A*, 161 586 1956
- Vandam L. D. and Dripps R. D. Exacerbation of pre existing neurologic disease after spinal anesthesia. *New England J. Med.*, 255 843, 1956

anesthesia, to the choice of the patient for this technique, and to the administration of the anesthetic as described

Exacerbation of Pre-existing Neurologic Disease after Spinal Anesthesia

Although a cause-and-effect relation between spinal anesthesia and exacerbation of disease can rarely be proved, it is our opinion that spinal anesthesia should not be given to a patient with affliction of the central nervous system or spinal column. This admonition applies to congenital, healed, inactive and active disease. It embraces trauma, bacterial and viral infections, degenerative diseases, neoplasms and systemic diseases with neurologic accompaniments. Exceptions to the rule may be made only when a type of anesthesia other than spinal is potentially more hazardous or less safe in the hands of the person administering it.

COMPARISON OF SPINAL AND EPIDURAL ANESTHESIA

A local anesthetic can be deposited in the space between the dura and the periosteum lining the vertebral canal. This is termed epidural anesthesia. No opening is made in the dura with this technique, so that sequelae related to changes in cerebrospinal fluid pressure are avoided. The chief advantage is elimination of post-lumbar puncture headache. The local anesthetic, although placed extradurally, does gain entry into the subarachnoid space, probably via the points of exit of spinal nerves and blood vessels. The spinal cord and subarachnoid space are thus exposed to a drug as part of epidural anesthesia, though to a lesser extent than with spinal anesthesia. The most recent modification of epidural anesthesia is the technique of intermittent injection of a local anesthetic through a catheter placed within the epidural space. With the tip of the catheter located at the point of exit of nerves to be anesthetized, a segmental block can be achieved, only that section of the body which is involved in the operation being anesthetized.

Studies of the neurologic sequelae of epidural techniques have not been as carefully performed as have those for spinal anesthesia, but the incidence of such complications probably would be less. Hypotension may be an accompaniment of any but limited epidural anesthesia. If the same spinal roots are blocked epidurally or in the subarachnoid space, the circulatory effect will be identical. Epidural anesthesia is technically more difficult to achieve and is therefore less reliable than spinal anesthesia. It is also slower in onset. For these reasons we do not believe that the method will ever replace subarachnoid block.

3 It is an ideal method for ambulatory patients, for short and superficial operations, and in situations where a recently ingested meal might pose a threat of vomiting during general anesthesia. If patient co-operation is needed, as, for example, in complicated tendon repairs of the forearm, local anesthesia is valuable.

Some of the reasons why local anesthesia is not more widely used may be listed as follows:

1 Lack of patient acceptance and the desire to be unconscious during operation. Part of this difficulty resides in an ineffectual approach to the patient and lack of skill in the performance of nerve blocks.

2 The impracticality of rendering some bodily areas anesthetic for operation. For example, the number of injections, the amount of anesthetic required, and the time consumed in providing local anesthesia for a radical mastectomy are prohibitive.

3 Inadequate duration of local anesthesia. There are, however, agents and special techniques that can prolong anesthesia.

4 Rapid absorption of local anesthetics into the blood stream. This may produce untoward reactions that can be detrimental to the patient. Although we understand the mechanism, means of prevention, and treatment, reactions still occur because of variations in human responsiveness.

LOCAL ANESTHETIC DRUGS

Cocaine, the first local anesthetic, is an ester of benzoic acid and the alcohol ecgonine. Although it is a potent topical and injectable substance with vasoconstrictive properties, the early high incidence of reactions, difficulty in sterilization, and lack of a synthetic product led to a search for a better anesthetic, particularly for infiltration. Investigations of ester combinations led to the synthesis of procaine by Einhorn in 1905. This is the ester of para-aminobenzoic acid and diethyl amino-ethanol. Procaine remains the standard for comparison with other anesthetics. Its popularity can be related to the low toxicity, ease of synthesis, low cost, ease of sterilization and the reversibility of local effects. The disadvantages are the lack of topical activity, and relatively short duration. Newer anesthetics must be assessed according to these criteria, particularly the properties of lack of irritation and that of reversibility to the preanesthetic state. Many so-called anesthetics have proven to be destructive to tissues and are not reversible in action.

Subsequent studies of anesthetics have indicated that the ability to

Fundamentals of Local Anesthesia

FOLLOWING the discovery by Koller in 1884 that cocaine could anesthetize the cornea, local anesthesia underwent rapid development. In the same year Halsted employed cocaine both for infiltration of the skin and direct nerve injection, while Corning attempted to anesthetize the spinal cord and nerves through an injection into the back. He performed what might have been an epidural injection. Surgeons became interested in local anesthesia because of dissatisfaction with general anesthesia and because they could give local anesthesia themselves. After the performance of lumbar puncture by Quincke in 1891 it was to be expected that someone would inject cocaine into the subarachnoid space. This was accomplished in 1899 by Bier and Tuffier independently. The subsequent development of local anesthesia lay in its application to regional nerve block and in the synthesis and study of new anesthetics.

Local anesthesia has continued to be popular for the following reasons:

1. It is easy to use local anesthetics. The method is economical, non-explosive, and the equipment required minimal. The need for postoperative observation and care of the patient is lessened.

2. Some of the undesirable side effects of general anesthesia are avoided. A localized area of the body can be operated upon without the loss of consciousness. Modern studies support the old anoci-association theory of Crile which held that impulses from the operative area could be harmful and lead to shock. Local anesthesia decreases the general stress response to trauma, perhaps through interference with afferent nerve impulses.

about by stabilization of electrical charges on the opposite sides of the surface membrane

Humoral Action

Acetylcholine has been postulated to be the transmitter substance in the mediation of the nerve impulse. It is said to be released from an inactive bound form, triggers the migration of sodium and potassium ions across the cell membrane, and is quickly hydrolyzed by cholinesterase. Thereafter acetylcholine assumes once more its bound state. Nachmansohn believes that these reactions take place within the time limits required for the passage of a nerve impulse. Local anesthetics may interrupt conduction through competition with acetylcholine at receptor sites.

Respiration of Nerve Fibers

Activity in the central and peripheral nervous systems is accompanied by the uptake of measurable amounts of oxygen, and the elimination of carbon dioxide. Local anesthetics may block conduction in nerve fibers without demonstrable changes in their oxygen consumption, however, it may be that the experimental methods available for this type of study are not sufficiently sensitive.

METABOLISM AND EXCRETION

Local anesthetic substances are inactivated mostly in the blood stream. Little destruction of ester compounds takes place in the cerebrospinal fluid. Esters are hydrolyzed in plasma by enzymes that also split acetylcholine. These enzymes, derived from the liver, are called pseudo-cholinesterases in distinction to the true cholinesterase found at the neuromyal junction. The rate of hydrolysis of procaine by cholinesterase in plasma has been shown to be in the order of one gram per hour for an adult of average size. In general the duration of local anesthesia will be prolonged if absorption from the injection site is slow and shortened if there is rapid destruction in the circulation. This is one of the reasons why epinephrine is used with local anesthetics—to decrease the rate of absorption.

The detoxification of non-ester substances is not understood. Small quantities of unchanged anesthetic are excreted in the urine without regard to basic chemical structure.

REACTIONS

Many laboratory experiments and observations in humans have demonstrated that reactions may be related to the blood concentration

produce local anesthesia need not be associated with an ester configuration. The use of dibucaine (Nupercaine) and more recently lidocaine (Xyllocaine) attests to this fact. Dibucaine is used largely for prolonged anesthesia. A greater toxicity is counterbalanced by the lesser concentrations required. Lidocaine has achieved wide usage recently because of the rapid onset of action, longer duration of action as compared to procaine, and because of an apparent tendency to spread from the site of injection.

Suspensions of anesthetics in oil have been advocated for long-lasting anesthesia. There is no evidence that a gradual release of the anesthetic from the menstruum is responsible for the reported long action. Rather, destructive substances like benzyl alcohol, incorporated in the mixture, can account for the prolongation of effect.

THEORIES OF ACTION

Most theories of local anesthetic action are those proposed for the action of general anesthetics. As with general anesthesia, there is no single acceptable explanation for the phenomenon of local anesthesia. A good way to summarize the problem is to relate anesthetic action to the known facts of transmission of the nerve impulse.

Electrical Activity

A nerve impulse can be described in terms of a predictable pattern of electrical disturbance. Local anesthetics abolish this discharge in a characteristic manner. Erlanger and Gasser, in studies of electrical potentials in mixed nerves, discovered that fibers of different size conducted impulses at different rates. The smaller nerve fibers, those subserving superficial pain and temperature, conducted slowly. It has been known for a long time that these small fibers are blocked first by local anesthetics, and it has been suggested that the greater surface area of the small fiber allows for more rapid penetration. An affirmation of this discovery is the observation that weak concentrations of local anesthetics interrupt conduction in small fibers first. This ability to block fibers differentially has become useful in diagnosis and therapy, as for example in differential subarachnoid block. In practice the concentration of local anesthetic chosen should be only that needed to block the desired nerve fibers. This is important in the prevention of reactions to local anesthetics since the total amount injected can thereby be decreased.

Local anesthetics also are said to block conduction in nerve fibers through interference with electrical depolarization. This is brought

lungs is as rapid as with intravenous injection and the path to the heart is a short one. An alternative possibility is the stimulation of a depressor chemoreflex of the von Bezold-Jarisch type.

Sensitivities and Allergic Phenomena

Dermatitis due to sensitivity to local anesthetics is not uncommon. Urticaria is seen occasionally but symptoms suggestive of true anaphylaxis are exceedingly rare. The latter comprise histamine release, bronchoconstriction, circulatory collapse, and failure of the blood to coagulate. These reactions can be minimized by careful query for drug sensitivity and other allergic responses. When a history of sensitivity is elicited, the substitution of an anesthetic with a totally different chemical configuration may avoid trouble. Skin wheals and patch tests are unreliable in predicting reactions. The common symptoms of tenseness, pallor, sweating, palpitation, dizziness, fainting, and sometimes anginal pain, are ordinarily the effects of epinephrine that has been added to the local anesthetic solution.

The phenomena in the first three categories are generally conceded to be due to an excessive amount of circulating local anesthetic. This means that the total quantity injected, the rapidity of absorption, and the rate of circulation, detoxification and excretion are the important factors. Since one cannot evaluate all these factors beforehand in addition to the unpredictability of patient susceptibility, the following routine is suggested for all patients who are to receive local anesthetics.

One hundred to 200 mg. of short-acting barbiturate given by mouth beforehand will allay apprehension, and may diminish the possibility of reaction. While there is laboratory evidence to show that barbiturates decrease the toxic effects of local anesthetics, the amounts that have been used in animals are closer to the anesthetic than to the sedative doses.

Operating room preparations should be the same as for any anesthetic procedure. The means for treating respiratory and circulatory complications must be at hand. A short-acting barbiturate should be given when signs of central stimulation have become evident, to avoid subsequent convulsion. The patient should be observed by an anesthetist throughout, engaged in conversation, and the circulatory and respiratory signs recorded.

One should try to estimate the duration of anesthesia needed and select a drug accordingly. Roughly, the range of action is one hour for procaine to four and five hours for tetracaine (Pontocaine), when epinephrine is used. Fresh, sterile local anesthetics should be chosen.

of local anesthetic The target organs for these reactions are the cerebrum, medulla, heart and peripheral blood vessels When the reaction is slow in development, stimulation of the cortex and medulla precedes a depressant phase Occasionally following the injection of even a minute amount of drug, respiration and circulation fail simultaneously without warning Local anesthetic reactions may be classified as follows

Central Nervous System Stimulation

During the course of anesthesia a patient should be observed for warning signs of a reaction to local anesthesia, and prompt preventive measures instituted should such signs appear Restlessness, talkativeness or muscle twitching may precede the onset of generalized convulsions The intravenous injection of a small dose of short-acting barbiturate (50 mg thiopental) and the administration of oxygen are important measures both in prevention and treatment

Central Nervous System Depression

Depression of the cerebral cortex leads to drowsiness, sleep or coma As mentioned above, this reaction may follow a stimulatory phase or may be the only central effect of certain anesthetics Failure of respiration and loss of central vasomotor control leading to hypotension indicate a depressant action on the medulla Severe depression can lead to simultaneous respiratory standstill and circulatory collapse Peripheral actions on sympathetic ganglia and direct vasodilation increase the hypotensive response This rapid collapse must be detected promptly and treated in the manner suggested for severe arterial hypotension

Peripheral Circulatory Depression

Local anesthetics depress the three properties of cardiac muscle contractility, irritability and rhythmicity These are the pharmacologic attributes that first led to the use of procaine and its derivative procaine amide for the treatment of cardiac arrhythmias Local anesthetics, in addition, interfere with synaptic transmission in the autonomic nervous system and also produce vasodilation through a direct vascular action The sum total of these actions diminishes cardiac output, impairs conduction in the heart, and may lead to arterial hypotension and cardiac arrest Direct cardiac depression is probably responsible for the high incidence of sudden vascular collapse seen when topical anesthetics are instilled into the trachea and bronchi Absorption from the

lungs is as rapid as with intravenous injection and the path to the heart is a short one. An alternative possibility is the stimulation of a depressor chemoreflex of the von Bezold-Jarisch type.

Sensitivities and Allergic Phenomena

Dermatitis due to sensitivity to local anesthetics is not uncommon. Urticaria is seen occasionally but symptoms suggestive of true anaphylaxis are exceedingly rare. The latter comprise histamine release, bronchoconstriction, circulatory collapse, and failure of the blood to coagulate. These reactions can be minimized by careful query for drug sensitivity and other allergic responses. When a history of sensitivity is elicited, the substitution of an anesthetic with a totally different chemical configuration may avoid trouble. Skin wheals and patch tests are unreliable in predicting reactions. The common symptoms of tenseness, pallor, sweating, palpitation, dizziness, fainting, and sometimes anginal pain, are ordinarily the effects of epinephrine that has been added to the local anesthetic solution.

The phenomena in the first three categories are generally conceded to be due to an excessive amount of circulating local anesthetic. This means that the total quantity injected, the rapidity of absorption, and the rate of circulation, detoxification and excretion are the important factors. Since one cannot evaluate all these factors beforehand in addition to the unpredictability of patient susceptibility, the following routine is suggested for all patients who are to receive local anesthetics.

One hundred to 200 mg. of short-acting barbiturate given by mouth beforehand will allay apprehension, and may diminish the possibility of reaction. While there is laboratory evidence to show that barbiturates decrease the toxic effects of local anesthetics, the amounts that have been used in animals are closer to the anesthetic than to the sedative doses.

Operating room preparations should be the same as for any anesthetic procedure. The means for treating respiratory and circulatory complications must be at hand. A short-acting barbiturate should be given when signs of central stimulation have become evident, to avoid subsequent convulsion. The patient should be observed by an anesthetist throughout, engaged in conversation, and the circulatory and respiratory signs recorded.

One should try to estimate the duration of anesthesia needed and select a drug accordingly. Roughly, the range of action is one hour for procaine to four and five hours for tetracaine (Pontocaine), when epinephrine is used. Fresh, sterile local anesthetics should be chosen

The concentration is determined by the character of the anesthesia needed. To reiterate, small nerve fibers, those that carry pain and temperature sensation, need only weak concentrations (procaine 0.5 per cent) for block, whereas larger fibers subserving muscle power, touch and position require greater concentrations (procaine 1.5 to 2.0 per cent). Using several concentrations and small total volumes, one should be able to avoid the upper limits of toxicity (procaine 0.5 to 1.0 gm).

Epinephrine when used to increase the duration of anesthesia and prevent rapid absorption of the anesthetic need not be employed in concentrations greater than 1:200,000. It should be measured in a syringe rather than counted in drops. Epinephrine carries hazards of its own in local vasoconstriction, central nervous system and cardiac stimulation as mentioned previously. If these are feared, other catecholamines such as Cobefrin or Kefrine may be used.

A small test dose should precede the injection of larger amounts of local anesthetic. Injection should be slow, as rapid injections are painful, spread the anesthetic, and sometimes may tear fragile, thin-walled blood vessels. One should aspirate before injecting a new area. If blood appears, the position of the needle should be changed before injection. The highest incidence of reactions occurs in vascular areas such as the head, face and neck, vertebral and paravertebral regions where absorption can be rapid. Severe pain, prolonged and unusual paresthesias, or other unexplained phenomena, should cause one to pause and seek explanation. The most successful local anesthetic procedures are artful ones, in which the patient is forewarned, encouraged, soothed, and the manipulations are slow and gentle. The operator must have a thorough knowledge of the anatomy of the area injected. Although infiltration of tissues is easy, the performance of specific nerve block requires considerable skill and experience. For the performance of nerve blocks we refer the reader to specialized texts for more detailed information.

REFERENCE

Moore, D. C. *Complications of Regional Anesthesia*. Charles C. Thomas, Springfield, Illinois, 1955.

III

During Operation

A Philosophy of Anesthetic Records

ANESTHETIC records can be of value to patient, anesthetist, surgeon and nursing staff. If one has at regular intervals observed and recorded the pulse rate, respiration, blood pressure and other pertinent factors, he can more accurately determine the patient's condition at a given moment. A good record of a previous anesthesia is of considerable help if a patient has to be anesthetized again. There are medico-legal reasons for keeping accurate records, since their review can establish the course of events more convincingly than recourse to memory. In an article entitled "Prevention of Malpractice Claims," Dillon states that in a ten year experience of reviewing cases of alleged malpractice, he has yet to see one in which the records of the anesthetic course were complete. If one engages in the simplest clinical research in anesthesia, records are a key aspect of data collection. Here it must be emphasized that conclusions drawn from such data are only as reliable as are the original, individual records.

A fine record is not much good if the anesthesia is poor, in other words, the patient should never be sacrificed for the record. There are times during difficult procedures when the patient demands complete attention. It would be foolish at such times to withdraw attention from him in order to complete the record. In the majority of cases, however, one should be able to keep a full and detailed account of the entire procedure.

An example of a suitable record is reproduced here. These should be kept in duplicate, one for the hospital chart, the other for anesthesia departmental filing and analysis.

*Data to be Provided on Anesthesia Records***Front of Chart***History number**Date* To avoid misunderstanding, record dates thus 25 Dec, '57*Ward* Write the ward to which the patient is to be returned after the operation. If the patient is an out-patient, write "OUT"

Hist. No. _____

Date _____

Ward _____

Name _____

Race _____ Sex _____

Address _____

Phone _____

Age _____

Ht _____

Wt _____

Hb _____

Recent

Meal _____

Phys. Exam _____

Preop Medication _____

Time _____

Oper. Perm. _____

Recent History _____

Drug Ther. _____

LP No. _____ Ga. gr _____

Tumors _____

INDUCTION & U _____

AIRWAY _____

E. S. S. _____

MAINTENANCE _____

Agent _____

Tech _____

Operation _____

Surgeon _____

Anesthetist _____

Position _____

Reflex recovery Time _____

Spont. Cloud

Circ. out. W. re

— Rich

Emme

TBT

— 2 ut MS

Total

FLUIDS

PSS

Cluc

Blood

Plasma

Name (Of patient) Last name first Print the last name and include the usual full name and titles

Address and telephone number (This information is important for postoperative follow-up studies) Enter the patient's permanent home address including postal zone

Age When a number is written alone it is understood to mean years If the age is measured in months, weeks or days, write thus 8 months, or 6 weeks, or 3 days

Ht Record the patient's height in inches If the height is not known, make an estimate

Wt Record the patient's weight in pounds If the weight is not known, make an estimate

Hb Record the hemoglobin in grams per 100 ml

Recent meal Ask the patient when he or she last ate or drank Enter "NO" if the patient has had nothing by mouth for at least six hours Otherwise write 'YES' and elaborate on the chart At the same time ask about removable teeth, chewing gum, or tobacco in the mouth

Remember Infants and small children usually have had a feeding within four hours of the time set for operation, injury, shock, emotional tension or excessive ingestion of alcohol almost always delays the emptying of the stomach, and, women in labor often have full stomachs

Physical status See Chapter 2 for numerical rating

Peanesthetic medication Write in the drugs—amount, time, route of administration and effect Note if sedation is adequate or if untoward effects on circulation, respiration or nervous system have occurred If no preliminary medication was given, write 'NONE'

Special information There should be a place at the top of every anesthesia record to write information of especial importance and to direct notice to it Thus, if a patient has a disease such as myasthenia gravis, active tuberculosis, or severe allergies, this information can be of vital importance

Operative permit Be certain that the patient has granted permission in writing for the performance of anesthesia and operation This is especially important for minors where the parent or guardian grants permission Individual states vary in their requirements and anesthesiologists must be familiar with the legal rulings in their communities

Neurologic history Here should be recorded the word negative if applicable, or reference should be made to any pertinent findings This is of value in medicolegal actions involving the anesthesiologist It is of particular importance if spinal anesthesia is to be given

Drug history As indicated in Chapter 20 many therapeutic substances

affect the course of anesthesia adversely Query and notation should be made for those drugs which the patient has been taking

L P This refers to spinal anesthesia In evaluating the causes of untoward sequelae of spinal anesthesia it is important to have some idea of the degree of trauma caused by the insertion of the lumbar puncture needle One should record the following the gauge of the lumbar puncture needle, the level of insertion of the needle on the spinal column, the type of lumbar puncture, whether midline or lateral, the number of needle insertions, the lack or production of paresthesias and their location, the appearance of the cerebrospinal fluid—whether clear or blood-stained, the ease of aspiration or flow of cerebrospinal fluid When using a catheter for serial spinal anesthesia, record the estimated length of catheter inserted beyond its entry into the sub-arachnoid space

Induction S U If the induction was satisfactory, circle the S If unsatisfactory, circle the U and state why Common causes for an unsatisfactory induction include emesis, retching, cough, soft tissue obstruction, laryngospasm, excess mucus, excitement, slow induction, apnea, respiratory depression, hypotension, cyanosis and pulse irregularities

Airway In this space are recorded the details of artificial airways used during anesthesia If a rubber or metal airway is passed through the mouth or the nose into the pharynx, write in "oropharyng" or "nasopharyng" Note should be made as to whether the insertion was atraumatic or accompanied by trauma such as bleeding, chipping of teeth, or injury to the lips

The same information regarding trauma is to be recorded when a tube is passed into the trachea In addition, "Endotracheal" should be circled and the following data supplied

Route of intubation orotracheal or nasotracheal, right or left

Number of attempts at intubation each insertion of a laryngoscope or each time the tube is passed beyond the epiglottis counts as an attempt When intubating blindly, each time the tube is advanced with the expectation or hope of its entering the larynx counts as an attempt

Size of tube, use of stylet

Method of intubation unless otherwise noted it is assumed that the intubation was performed under direct vision with a laryngoscope If the tube was inserted without benefit of laryngoscope, write in "Blind"

Unless otherwise noted it is assumed that the tube is used simply as an airway under an open or closed mask When the tube is used

without a mask, when it is connected more or less directly to a bag, canister, delivery tube or Y tube, note if the tube is cuffed (note separately when cuff is inflated), or if a pack was placed in the pharynx.

Maintenance This space is for recording interesting or important remarks, facts or events which occur during the anesthesia and operation. It is impossible to list all such events, and in any case their significance varies with the particular circumstances. As experience increases, one will become better able to select what should be recorded and what should be omitted.

Record facts about the condition of the patient other than the pulse, respiration and blood pressure. These include coughing, pallor, sweating, and the character of the pulse or respiration if it is abnormal, the presence of excessive secretions, laryngospasm, hiccough, fever, tremor, twitching and convulsions. Record also facts concerning the conduct of the anesthesia which cannot be shown on the graphic chart, e.g., the presence of respiratory obstruction, of oxygen want, the introduction or removal of the carbon dioxide absorber from the system, the wearing off of spinal or regional block anesthesia. It is important to note the reasons for changing anesthetic agents or methods and to record the time at which the patient's position is changed, since alterations in respiration or circulation often follow such a change.

Treatment used during the procedure which is not recorded elsewhere should be noted in this space. An example is tracheobronchial toilet (usually abbreviated 'TBT') which means the aspiration of mucus or other material from the trachea or bronchi by means of suction with a catheter inserted through the vocal cords or through an endotracheal tube. The amount and route of administration of drugs should be recorded. If blood is given, a positive identification of each bottle must be noted here.

Facts concerning the surgical procedure which may be of significance in regard to the condition of the patient or to the conduct of anesthesia should be written. Traction in the region of the gallbladder may produce changes in blood pressure. Opening the pleural cavity usually alters the character of the respiration. Clamping of the pulmonary artery may influence the cardiovascular system. The possibilities are legion. It is important to note the time when these events occur and to relate them to the progress of the procedure as recorded on the graphic chart. The simplest method is to number the notations 1, 2, 3, etc., and to write a similar number below the lower border of the graphic chart at the proper time scale position.

Agents Record all anesthetic and analgesic agents and adjuvants

used. The amount or strength of the agent must be included even in the case of agents administered by inhalation. In general the main agent is the one with which the greatest depth of anesthesia is obtained (except when a potent agent has been used only for induction). When thiopental is used with nitrous oxide-oxygen, thiopental is considered the main agent even though the greater part of the maintenance may have been accomplished by the nitrous oxide. If, in such a case, ether is added to produce greater relaxation, ether is considered the main agent. A basal narcotic such as tribromethanol (Avertin) should never be considered the main agent unless the entire operation has been performed under its influence without the addition of other agents, but anesthetics used for basal narcosis must be recorded.

When spinal or other regional analgesia is used with the idea of performing the scheduled operation under its influence alone, it should be considered the main agent even though the spinal or block fails and another agent has to be given, however, the other agent must be listed also. In the case of spinals, record the total volume of fluid injected as well as the name and amount of each drug used.

Technique Record the technique or method of administration of anesthetic agent. Write the technique by which a particular agent is given directly below the name of the agent.

<i>Agents</i>	Thiopental 2.5/	Ether
<i>Technique</i>	I V	Abs to and fro

Definitions of techniques The following methods and abbreviations are obvious:

- Intravenous—I V
- Subcutaneous—H or S C
- Infiltration—Infilt
- Block—specify
- Spinal—Sp, meaning 'single dose' spinal
- Serial spinal—Ser Sp
- Topical—Top
- Rectal—Rect

The various inhalational techniques are more difficult to classify. More than one method may be used for the same agent during the same anesthesia. Changes in method should be recorded.

without a mask, when it is connected more or less directly to a bag, canister, delivery tube or Y tube, note if the tube is cuffed (note separately when cuff is inflated), or if a pack was placed in the pharynx

Maintenance This space is for recording interesting or important remarks, facts or events which occur during the anesthesia and operation. It is impossible to list all such events, and in any case their significance varies with the particular circumstances. As experience increases, one will become better able to select what should be recorded and what should be omitted.

Record facts about the condition of the patient other than the pulse, respiration and blood pressure. These include coughing, pallor, sweating, and the character of the pulse or respiration if it is abnormal, the presence of excessive secretions, laryngospasm, hiccough, fever, tremor, twitching and convulsions. Record also facts concerning the conduct of the anesthesia which cannot be shown on the graphic chart, e.g., the presence of respiratory obstruction, of oxygen want, the introduction or removal of the carbon dioxide absorber from the system, the wearing off of spinal or regional block anesthesia. It is important to note the reasons for changing anesthetic agents or methods and to record the time at which the patient's position is changed, since alterations in respiration or circulation often follow such a change.

Treatment used during the procedure which is not recorded elsewhere should be noted in this space. An example is tracheobronchial toilet (usually abbreviated "TBT") which means the aspiration of mucus or other material from the trachea or bronchi by means of suction with a catheter inserted through the vocal cords or through an endotracheal tube. The amount and route of administration of drugs should be recorded. If blood is given, a positive identification of each bottle must be noted here.

Facts concerning the surgical procedure which may be of significance in regard to the condition of the patient or to the conduct of anesthesia should be written. Traction in the region of the gallbladder may produce changes in blood pressure. Opening the pleural cavity usually alters the character of the respiration. Clamping of the pulmonary artery may influence the cardiovascular system. The possibilities are legion. It is important to note the time when these events occur and to relate them to the progress of the procedure as recorded on the graphic chart. The simplest method is to number the notations 1, 2, 3, etc., and to write a similar number below the lower border of the graphic chart at the proper time scale position.

Agents Record all anesthetic and analgesic agents and adjuvants

CONTROLLED RESPIRATION—this is the situation where the anesthetist "breathes" for the patient, controlling both rate and depth of respiration, after a state of physiologic or pharmacologic apnea has been produced. Apnea may be produced by one or more of the following means: hyperventilation, the administration of drugs which depress the activity of the respiratory center, or the administration of muscle relaxants.

ASSISTED RESPIRATION—this is often used instead of controlled respiration to minimize movements of the mediastinum, the diaphragm and respiratory muscles. The patient continues to make respiratory efforts but the anesthetist applies pressure just before or during inspiration. Pressure is not applied during expiration.

Operation Write the complete and accurate name of the operative procedure. If in doubt, ask the surgeon to name the operation.

Surgeons Write the name of the surgeon who performs the operation. If in doubt as to who the surgeon is, ask, as this may be important for future reference. The name of the assistants should be included. Include initials if necessary to prevent confusion.

Anesthetists Write the names of the anesthetist and assistants concerned.

Instr Write here the name of the instrument nurse.

Position Fill in the appropriate term for the positions of the patient during operation.

Sponge count The anesthetist may write on this line, or the responsible nurse may sign her name.

Recovery—Reflex in O R Record the condition of the patient on leaving the operating room, in regard to recovery of consciousness and reflex activity.

Fluids Write the amounts of fluids given during anesthesia on the appropriate lines.

The Chart or Graph

Vertical lines time

Horizontal lines the chart may be divided horizontally into several sections, from above downward:

- 1 Anesthetic agents
- 2 Depth of anesthesia
- 3 Sensory level of spinal anesthesia, as determined by pin prick
- 4 Graph for the record of blood pressure, pulse rate, respiratory rate

The open and insufflation techniques are generally used without a breathing bag or other respiratory reservoir and without a carbon dioxide absorber. There are, however, exceptions.

OPEN—a system is open only when there is free escape of expired air to the outside and when rebreathing is impossible. The Leigh and Stephen-Slater valves permit this type of gas exchange.

SEMI-OPEN—the line between semi-open and semi-closed may be difficult to distinguish, but "semi-open" is the proper term when an open mask is surrounded or covered with towels or gauze so that the free escape of carbon dioxide is hindered.

INSUFFLATION—the gas or vapor is delivered to the mouth, pharynx or trachea and escapes by a path other than that by which it enters the patient. When insufflation is performed with the aid of an endotracheal tube, in order to provide for expiration, an Ayre or a Y tube is usually placed between the endotracheal tube and the tube delivering the gas or vapor.

FLAGG CAN—this simple method does not readily fit into any one of these categories. It is usually employed with an endotracheal tube so that practically the whole of the respiratory exchange passes through the apparatus. The increased dead space can therefore be considerable.

SEMI-CLOSED—this denotes the method in which the patient inspires from a system with a breathing bag into which gas or vapor or both are flowing at a high rate (4 to 7 liters per minute) and in which there is a valve or other means of overflow so that during expiration a portion of the expired gases escape. There is therefore some rebreathing.

CLOSED—this implies that the ambient atmosphere is excluded from the respiratory exchange and that none of the gas in the system is discharged into the atmosphere. A minor leak around the face mask does not preclude calling the technique "closed."

NO ABSORPTION—carbon dioxide frequently is not absorbed during the induction of anesthesia but to use such a technique for more than 3 or 4 minutes is unwise. (Three or 4 minutes without carbon dioxide absorption will lead to the accumulation of about 3 to 4 per cent carbon dioxide in inspired air.) Whenever the closed technique is used without absorption, close attention must be paid and the patient's condition must be assessed more frequently than usual. Patients vary in their sensitivity to carbon dioxide according to the degree of preanesthetic depression, the anesthetic agents and the depth of anesthesia.

ABSORPTION—the use of an absorber of carbon dioxide must be qualified by either "to-and-fro" or "circle."

CONTROLLED RESPIRATION—this is the situation where the anesthetist "breathes" for the patient, controlling both rate and depth of respiration, after a state of physiologic or pharmacologic apnea has been produced. Apnea may be produced by one or more of the following means: hyperventilation, the administration of drugs which depress the activity of the respiratory center, or the administration of muscle relaxants.

ASSISTED RESPIRATION—this is often used instead of controlled respiration to minimize movements of the mediastinum, the diaphragm and respiratory muscles. The patient continues to make respiratory efforts but the anesthetist applies pressure just before or during inspiration. Pressure is not applied during expiration.

Operation Write the complete and accurate name of the operative procedure. If in doubt, ask the surgeon to name the operation.

Surgeons Write the name of the surgeon who performs the operation. If in doubt as to who the surgeon is, ask, as this may be important for future reference. The name of the assistants should be included. Include initials if necessary to prevent confusion.

Anesthetists Write the names of the anesthetist and assistants concerned.

Instr Write here the name of the instrument nurse.

Position Fill in the appropriate term for the positions of the patient during operation.

Sponge count The anesthetist may write on this line, or the responsible nurse may sign her name.

Recovery—Reflex in O R Record the condition of the patient on leaving the operating room, in regard to recovery of consciousness and reflex activity.

Fluids Write the amounts of fluids given during anesthesia on the appropriate lines.

The Chart or Graph

Vertical lines time

Horizontal lines the chart may be divided horizontally into several sections, from above downward:

- 1 Anesthetic agents
- 2 Depth of anesthesia
- 3 Sensory level of spinal anesthesia, as determined by pin prick
- 4 Graph for the record of blood pressure, pulse rate, respiratory rate

The open and insufflation techniques are generally used without a breathing bag or other respiratory reservoir and without a carbon dioxide absorber. There are, however, exceptions.

OPEN—a system is open only when there is free escape of expired air to the outside and when rebreathing is impossible. The Leigh and Stephen-Slater valves permit this type of gas exchange.

SEMI-OPEN—the line between semi-open and semi-closed may be difficult to distinguish, but "semi-open" is the proper term when an open mask is surrounded or covered with towels or gauze so that the free escape of carbon dioxide is hindered.

INSUFFLATION—the gas or vapor is delivered to the mouth, pharynx or trachea and escapes by a path other than that by which it enters the patient. When insufflation is performed with the aid of an endotracheal tube, in order to provide for expiration, an Ayre or a Y tube is usually placed between the endotracheal tube and the tube delivering the gas or vapor.

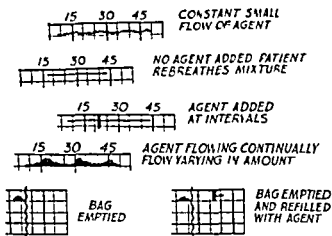
FLAGG CAN—this simple method does not readily fit into any one of these categories. It is usually employed with an endotracheal tube so that practically the whole of the respiratory exchange passes through the apparatus. The increased dead space can therefore be considerable.

SEMI-CLOSED—this denotes the method in which the patient inspires from a system with a breathing bag into which gas or vapor or both are flowing at a high rate (4 to 7 liters per minute) and in which there is a valve or other means of overflow so that during expiration a portion of the expired gases escape. There is therefore some rebreathing.

CLOSED—this implies that the ambient atmosphere is excluded from the respiratory exchange and that none of the gas in the system is discharged into the atmosphere. A minor leak around the face mask does not preclude calling the technique 'closed'.

NO ABSORPTION—carbon dioxide frequently is not absorbed during the induction of anesthesia but to use such a technique for more than 3 or 4 minutes is unwise. (Three or 4 minutes without carbon dioxide absorption will lead to the accumulation of about 3 to 4 per cent carbon dioxide in inspired air.) Whenever the closed technique is used without absorption, close attention must be paid and the patient's condition must be assessed more frequently than usual. Patients vary in their sensitivity to carbon dioxide according to the degree of preanesthetic depression, the anesthetic agents and the depth of anesthesia.

ABSORPTION—the use of an absorber of carbon dioxide must be qualified by either "to-and-fro" or "circle."



2 DEPTH OF ANESTHESIA When a patient is anesthetized the estimated level of narcosis should be charted in the space labeled "Plane of 3rd Stage" every time the vital signs are recorded

3 LEVEL OF SPINAL ANESTHESIA A space is provided in which to write the level of sensory block. These levels should be determined as frequently as practicable, but especially at the beginning and end of the procedure

4 PULSE RATE, BLOOD PRESSURE AND RESPIRATORY RATE These are to be charted on the larger portion of the graph. A solid dot is used for pulse rate, an open circle for respiratory rate, a ∇ for systolic and \wedge for diastolic pressure

The pulse, respiratory rate and blood pressure should be determined and recorded at least every five minutes (See Chapter 8)

Along the bottom of the graph mark the symbols denoting the start and end of the anesthesia and of operation

Start of anesthesia — \times

Start of operation — \bigcirc

End of anesthesia and operation — \otimes

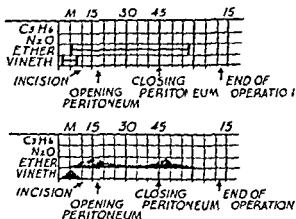
Back of Chart

The back of an anesthetic record should provide space for a summary of the preoperative condition as well as space for postoperative notes. A conscientious analysis and recording of every significant event in a patient's postoperative course can provide valuable data. It is common experience to hear expressions of opinions as to the incidence or severity of a particular complication. The fallibility of such statements is obvious. Unless reliable data have been recorded and studied such beliefs deserve little attention. If each patient's chart contained an

1 ANESTHETIC AGENTS In this section there are spaces for recording the administration of various anesthetic agents

The graphic recording of intravenous or intrathecal agents is fairly obvious. These are usually given in measured doses at separate intervals. With serial spinal anesthesia one should make a mark in the appropriate space each time the drug is given, and record the amount of the dose.

When the open method for administration of inhalation agents is used, the agent is generally administered more or less constantly throughout the procedure. The examples below illustrate how one might record a vinethene-ether anesthesia by the open technique. The remarks and arrows leading from them have been added for general information. On an actual chart these remarks would not be recorded in this manner.



The first example is the simpler of the two. In the second example an attempt has been made to record the relative rates at which the agents were administered at different times.

The semi-closed technique would be recorded in the same fashion. The recording of the closed technique is a little different. As long as the mixture of gases does not escape from the circuit, the patient continues to breathe the anesthetic agent. During maintenance some anesthesiologists keep a constant small flow of the agent running all the time, others add to the mixture at intervals. In addition, at times, through accident or design the breathing bag is emptied. All these maneuvers should be recorded. For the sake of clarity and uniformity the following code should be used.

Disturbances in Motor Activity Paresis (partial paralysis) or complete paralysis should be recorded, including all evidences (subjective or objective) of weakness, changes in gait, and abnormalities of bowel and bladder function. These should be described accurately in accordance with accepted neurologic terms. Patients to whom spinal anesthesia has been administered should be directly questioned for these complications.

Mental State One of the hazards of general anesthesia is the development of psychosis. Aged individuals are apt to show mental disturbances of varying degree in the first few postoperative days. Disorientation may also occur as the result of narcotics other than anesthetics, a reaction common in the aged. Emergence delirium and excitation in the immediate postoperative period should be recorded in detail unless the recovery room record is appended. The therapy used for such excitation should be noted. Delayed recovery of consciousness and the occurrence of convulsions should be listed.

Respiratory Tract

Complications can be grouped together on an anatomic basis, i.e., location in the respiratory passages. Thus results of tracheal intubation may be included with atelectasis and pneumonia.

Sequelae of Tracheal Intubation These include delayed evidence of trauma to nose, mouth, pharynx and larynx. One should record edema, obstruction to respiration, local infection, subcutaneous emphysema, mediastinal emphysema (from injury to respiratory mucosa with tube or laryngoscope), hoarseness, "sore throat," cough (see Chapter 13).

Major Respiratory Complications Atelectasis or bronchopneumonia (this diagnosis is made if the clinical course is prolonged, the febrile reaction decreases only slowly, toxemia is evident, and there are roentgenographic signs of consolidation). Miscellaneous complications include hiccough, pleurisy, pneumothorax, aspiration pneumonia. Pulmonary edema and pulmonary embolus are recorded under circulation.

Circulatory System

Obvious complications such as shock, hemorrhage, cardiac arrhythmias, thrombophlebitis and pulmonary embolus should be recorded. Sequelae related to the anesthetic management of the patient such as low blood pressure and bradycardia following recovery from cyclopropane, and prolonged hypertension and/or tachycardia related to pressor drugs, should also be noted.

If acute cardiac failure and pulmonary edema occur, one should

accurate summary of the convalescence, one would be in a position to state with reasonable certainty that (at least as far as experience in a particular clinic is concerned) the incidence of postural headache following spinal anesthesia was ——— per cent, or that vomiting occurred in ——— per cent of patients receiving cyclopropane

The source of the details includes the patient, the nursing notes, the surgeon's notes and one's own observations. Every patient should be seen during the first 24 hours after operation and again on the second, fourth and sixth postoperative days. The frequency and duration of visits depend upon the patient's condition. Sufficiently close contact should be maintained during the patient's entire stay in the hospital to enable the anesthetist to record delayed phenomena such as pulmonary embolism, wound disruption, or a postlumbar puncture headache which might begin on the seventh to tenth postoperative day. The number of days during which the patient was followed should be recorded.

Although the actual form of the postoperative chart may vary, there are certain fundamentals to which the anesthetist should adhere as he records the anesthetic and surgical convalescence. These will be described according to systems.

Central Nervous System

Headache An adequate description of a headache includes the following: Date of onset, date out of bed (to analyze relationship to early ambulation), duration, location—i.e., frontal, occipital, nuchal, etc., severity—mild, moderate or severe, psychologic or emotional make-up of patient, presence of nausea, vomiting, stiff neck or dizziness, relation to posture—a nonpostural postspinal headache with stiff neck suggests a meningeal irritative reaction, disturbances of hearing or vision. Dimness, blurring of vision or diplopia should be noted. 'Blocking of the ears,' diminution in hearing and tinnitus may be related to changes in cerebrospinal fluid pressure communicated to the internal ear.

Disturbances of Sensation Under this heading one should list hyperesthesia and hypoesthesia, i.e., increased or decreased perception of any sensory modality (touch, pain, vibration, temperature and position). Patients who have received spinal anesthesia should be specifically questioned for the presence of numbness, tingling or pain particularly in the perineum or lower extremities. The question can be put casually but the answer should be recorded in each case. Backache can be recorded under the heading of sensory disturbance.

Determination of the Depth of General Anesthesia

WITH THE introduction of potent anesthetic agents physicians recognized the necessity of observing the reactions of the patient to the drug lest an overdose be given. During general anesthesia the problem is twofold: to determine the level or depth of anesthesia, and to judge what level is best for each patient from moment to moment. Presumably, the less drug the better, but enough drug must be given to provide optimal conditions for operation consistent with the patient's safety. It is not easy to determine where the line should be drawn.

Determination of the depth of anesthesia is important for a number of reasons:

- 1 The novice in anesthesia must have signposts along the path of increasing narcosis. Otherwise he is hopelessly lost as he begins the administration of anesthesia.

- 2 There is greater safety for the patient if one can determine the depth of anesthesia with reasonable accuracy.

- 3 A number of physiologic responses are blocked at certain levels of anesthesia. One must be able to know when such interruption of function has taken place, at least one should have a reasonable idea so that if desirable this state can be attained, if undesirable it can be avoided. Examples of such functions include relaxation of the anterior abdominal muscles, and relaxation of the pregnant uterus.

- 4 If an investigator is to confirm or extend another's observations, he must be able to duplicate experimental conditions. Both sets of experiments should be conducted under comparable circumstances—

evaluate the role of parenteral fluid therapy, hypoxia, respiratory obstruction, hypertension or hypotension as causative factors. If signs of coronary insufficiency (angina or infarction) develop a similar evaluation should be charted.

Gastrointestinal Tract

Nausea and Vomiting Although it is difficult to weigh the significance of a particular cause of nausea or vomiting in the postoperative period, a start can be made in this direction if certain details are recorded. These are the time of onset, duration and severity. For nausea alone, indicate as mild, moderate or severe, for vomiting indicate the number of times a patient vomited per 24 hours, the presence of gastrointestinal drainage, the relation to administration of opiates, and the previous history of such sequelae.

Abdominal Distention One is interested in the same factors as in the case of nausea and vomiting.

Liver

Record any degree of liver damage and evaluate the role of anesthetic management in its production.

Kidney and Bladder

The prime concerns are oliguria, anuria and urinary retention. Hypotension, incompatible transfusions and drugs should be evaluated as possible causes. The therapy employed for such sequelae is of interest. The nature of retention can best be stated by recording the number of catheterizations per 24 hour periods. If an indwelling catheter or suprapubic tube is in place, so state on the chart.

FOLLOW-UP NOTES ON HOSPITAL CHART

In addition to the data written on the back of the clinical anesthesia record, follow-up notes should be made on the patient's permanent hospital record. Significant facts concerning the patient's postoperative course are recorded over the signature of the anesthetist. Headache, pulmonary, cardiovascular, gastrointestinal and urinary tract complications should be analyzed from the standpoint of the etiologic role of anesthesia.

REFERENCES

- Beecher, H. K. The first anesthesia records. *Surg. Gynec. & Obst.* 71:689 1940.
 Dillon, J. B. Prevention of claims for malpractice. *Anesthesiology* 18:Sept. 1957.
 Waters, R. M. The teaching value of records. *J. Indiana M. A.* 29:110 1936.

to the loss of consciousness. Those parts of the brain of most recent phylogenetic development appear to be depressed first. This results in obtundation of intellect, memory, integrative functions and perception of time and space. Although this is commonly called the stage of analgesia, the sensation of pain is not absent and the pain threshold is apparently unchanged, but the patient's reaction to pain is altered. If warned, the patient will usually tolerate procedures that he normally would not—that is, minor surgical procedures and the second stage of labor. Recent studies suggest that analgesia is more profound with emergence into the first stage from deeper levels of anesthesia rather than during induction. Unfortunately, no objective sign has yet been found to indicate the transition to Stage II in which stimuli may cause the patient to react violently. Kaye states that the response to stroking of the eyelashes disappears at the bottom of Stage I. This might be used as a guide.

With thiopental, Stage I is a stage of amnesia rather than analgesia. The patient will react, often violently, to minor surgical procedures but will usually not remember such reactions. This is an indication of the lack of adequacy of thiopental as an anesthetic agent, and is one of the reasons for the use of supplemental nitrous oxide or opiates (see p. 87).

Stage II, the stage of delirium, lasts from the loss of consciousness to the onset of automatic breathing and disappearance of the lid reflex, here meaning the normal attempt of the eyelids to close when forcibly opened. This is a stage of unconsciousness with uninhibited action. Patients should not be stimulated in this stage, as responses are uncontrolled. Respiration may be irregular, the pupils are dilated but react to light. The pharyngeal and laryngeal reflexes are obtunded at the lower limits of this stage, although vomiting can occur through upper Plane I of Stage III. The chief reason for rapidly increasing the inhaled concentration of anesthetic is to pass through this stage quickly.

With thiopental, except for the alcoholic, muscular or apprehensive individual, this stage is seldom seen. It may also be absent with cyclopropane, chloroform and ethyl chloride.

Stage III, the stage of surgical anesthesia, lasts from the onset of automatic breathing to the cessation of respiration. Most surgical procedures are performed at this level. There are those who believe that it is dangerous to begin surgical procedures before third stage anesthesia has been reached. As John Snow put it, 'The surgeon wishes to know whether the patient will lie still under the knife.' For more accurate estimation of level this stage is subdivided into four parts.

Plane I is entered when the lid reflex is abolished and respiration

in this case at the same depth of anesthesia. Means for describing and relating anesthetic levels are therefore essential in research.

It would be useful to be able to determine the depth of anesthesia exactly in every patient at every moment. We are unfortunately far from this goal, and must therefore resort to estimates. Today, these are made on the basis of several criteria:

Clinical Observation. The "signs" of anesthesia recorded by observant clinicians have been grouped together into four "stages." They require no special instruments for detection. They are reactions on the part of the patient which are present for the alert anesthetist to observe. As has been remarked, the patient "talks to you with everything but his voice—with his eyes, pulse, chest, and abdomen. You must listen and interpret."

The Electroencephalogram. This is a specialized technique and requires expensive equipment. It is readily applicable clinically, however.

The Concentration of Anesthetic in Arterial Blood or End-Expiratory Gas. These are laboratory techniques. The results become available minutes to hours after the samples have been obtained.

The "signs and stages" will be described in detail since they constitute the most available means for assessing the depth of anesthesia. We believe that the future holds promise in improved monitoring of a patient's response to anesthetics through the use of objective measurements. Possibilities along this line will therefore also be considered.

SIGNS AND STAGES OF GENERAL ANESTHESIA

The division of anesthesia into stages was recognized soon after Morton's demonstration. In 1847 Plomley described three stages and later that year John Snow added the fourth stage, that of overdose. But little was written about the matter until Guedel, faced with the problem of training large numbers of technicians in the First World War, more closely defined and described the signs and stages of anesthesia. This work of Guedel's has been accepted the world over and is considered one of the most important contributions to anesthesia. These signs and stages will be seen best with those agents that provide a slow induction and gradual increase in the blood concentration. Thus they apply chiefly to ether anesthesia. Gillespie in 1943 pointed out that depth of anesthesia could be ascertained through study of reflex action, that is, the manner in which respiration, muscle tone and various reflexes are modified during the deepening of anesthesia. The signs to be discussed emphasize these changes.

Stage I is defined as lasting from the beginning of the anesthetic

Pupillary signs are less reliable over the age of fifty. The best attitude is to regard dilated pupils as a sign of overdose (save in second stage) until it can be proved otherwise. Lack of oxygen will also dilate the pupils. Indeed this may be the cause of dilated pupils in deep anesthesia.

Reflex closing of the vocal cords, or laryngospasm, begins to disappear in Plane 2. The secretion of tears gradually decreases in this plane, though with cyclopropane lacrimation may persist longer. The respiratory response to a skin incision disappears.

Muscular tone lessens as anesthesia deepens, but this is not always a reliable measure of the depth of anesthesia. Muscular tone varies enormously in individuals, as does the apparent flaccidity of the peritoneum. These are influenced by age, constitution, the degree of intestinal distention and the friability of tissues. The tonus of a flaccid muscle may be increased by a stimulus. Thus, a gentle surgeon will obtain as good abdominal exposure in second plane as a rough surgeon will in third plane. Oxygen lack or carbon dioxide excess may also cause muscle rigidity.

Plane 3 is entered when intercostal activity begins to decrease. Complete intercostal paralysis occurs in lower Plane 3 and respiration is carried on solely by the diaphragm. As a consequence, the depth of respiration is reduced. Inspiration is now shorter than expiration, and the pause between is longer than in lighter planes of anesthesia.

Another result of intercostal paralysis is the production of excessive diaphragmatic movement. This can be most annoying during intra-abdominal operations and may lead the surgeon to ask in error to 'get the patient deeper'. If anesthesia is lightened so that the intercostals function, diaphragmatic movements will usually decrease. The same result can be accomplished by assisting the respiration so that the diaphragm will not have to work so hard. In robust patients the latter is often the better course, for the surgeon may need considerable abdominal relaxation. Mushin has pointed out that if the intercostals are paralyzed the abdominal muscles will also be paralyzed, since the two sets of muscles are morphologically the same and have the same nerve supply.

During general anesthesia the upper intercostal muscles become paralyzed before the lower, this is fortunate because the anesthetist can usually feel the upper intercostal area and take warning. Contraction of the intercostal muscles lags behind that of the diaphragm. To detect this lag one must observe both diaphragmatic and intercostal activity and establish their time relationship. When the intercostals become

becomes regular. During this plane the duration of inspiration is usually longer than expiration, though this may be difficult to detect. With ether or nitrous oxide respiration is usually deeper than normal. With thiopental this is rarely the case, and seldom is it true with cyclopropane or chloroform. Respiration is regular with these agents, but tends to be more shallow and slower than normal, especially if the pre-anesthetic medication has included a narcotic. Without such medication respiration is more active. The respiratory rate will usually increase with painful stimulation.

In Plane 1, the eyeballs usually oscillate but may become eccentrically fixed. The movement may not be apparent immediately as the lids are drawn back, especially if only one eye is inspected. It is better to examine both simultaneously. The movements may cease if the lids are kept open too long, but will return after they have been closed for about 30 seconds. It is helpful to remember that if the eye muscles are tonic and active the abdominal muscles are probably in the same condition.

During Plane 1 the vomiting reflex is gradually abolished. It is curious that swallowing, retching and vomiting reflexes disappear in that order during induction, and reappear in the same order during emergence from anesthesia. The reason for this is not clear.

The secretion of tears increases through Plane 1, and the respiratory response to a skin incision decreases. In lower Plane 1 reflex closing of the eyelids, when the conjunctiva is stimulated, disappears. However, we decry traumatic methods of testing reflex responses such as touching the cornea.

Plane 2 lasts from the time the eyeballs cease moving and become concentrically fixed to the beginning of a decrease in the activity of intercostal muscles. Although this is true with diethyl ether, not infrequently with cyclopropane or divinyl ether the eyeballs will not be concentrically fixed although all other signs indicate that anesthesia is at the level of Plane 2 or 3.

Respiration is still regular, but tidal volume is diminished. The duration of inspiration and expiration may be equal or expiration may be slightly prolonged. With cyclopropane or an intravenous barbiturate, respiration will be more shallow than with ether and the impression is created that anesthesia is deeper than it really is.

The pupils may begin to dilate in Plane 2 with diethyl ether anesthesia. On the whole, pupillary size is an unreliable guide in patients given preanesthetic medication. Morphine tends to constrict the pupils while belladonna drugs produce dilatation. The effects of morphine generally overshadow those of atropine or scopolamine.

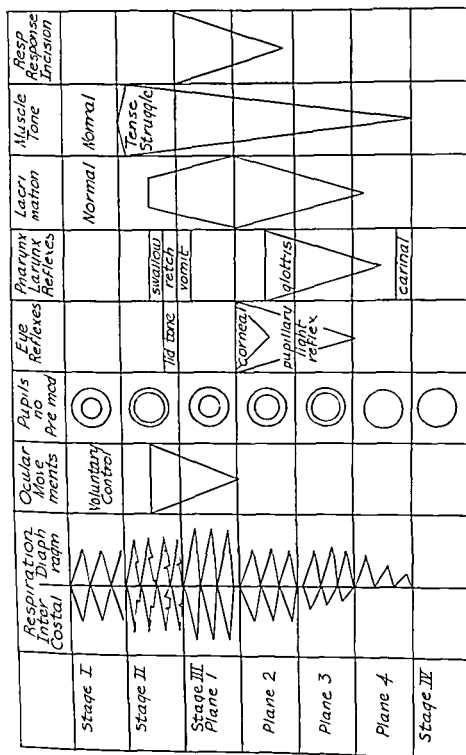


Fig 29 The signs and reflex reactions of the stages of anesthesia (After Gillespie Current Researches in Anesth & Analg 22 275-282 1943)

paralyzed, passive retraction of the chest on inspiration usually occurs and can give a false impression of intercostal activity unless the time relationship is carefully observed

It is unwise and rarely necessary to maintain Plane 3 for very long. Once the surgeon has explored the abdomen, obtained satisfactory exposure, and mobilized the organs, anesthesia can usually be lightened to upper Plane 2 or lower Plane 1.

In mid to lower Plane 3, the reactivity of the pupil to light is lost. With cyclopropane the pupillary light response may be lost in the second plane. The secretion of tears is steadily reduced as anesthesia deepens, but may persist well into Plane 3, especially with cyclopropane.

Plane 4 lasts from the time of paralysis of the intercostal muscles to the cessation of spontaneous respiration. As anesthesia deepens through this plane, diaphragmatic activity and respiratory exchange become progressively reduced until breathing stops. The pupils no longer react to light and dilate. There is little muscle tone even in a robust individual.

Tracheal tug often appears to be associated with deep anesthesia and intercostal paralysis. Waters states that it is seen also with accumulation of carbon dioxide. There is no satisfactory explanation for tracheal tug. It may be due to an attempt by the muscles in the neck to augment respiration.

Stage IV lasts from the time of cessation of respiration to failure of the circulation. It is entered when respiration fails consequent upon the increased concentration of anesthetic in the central nervous system. This is not to be confused with apnea due to breath-holding. Nor should it be confused with the reflex apnea sometimes seen in the third stage associated with manipulation of thoracic or abdominal structures, with periosteal, or with pharyngeal, laryngeal or bronchial irritation.

In Stage IV most reflexes are absent and the circulation is about to fail. The fourth stage is a premortem stage, and is of academic interest only. This plane of anesthesia should be produced only by mistake. Prompt steps should be taken to lighten the anesthesia, since even brief exposure to this plane usually leads to circulatory collapse. The concentration of anesthetic in the patient's circulation should be lowered by manual ventilation of the lungs with oxygen until spontaneous respiration resumes.

Finally, it must be remembered that anesthesia almost invariably appears to be deeper than it actually is. Since it is easier to lighten anesthesia than to deepen it, it is advisable to have a small margin of

example, influences the tracing. A lower concentration of ether is required to produce a given electroencephalographic level when nitrous oxide is present in the arterial blood in excess of 10 mg /100 ml. Carbon dioxide accumulation also affects the electrical pattern obtained from the brain. At a given arterial blood concentration of ether, the addition of carbon dioxide will produce an electroencephalographic level indicative of greater depression of the brain than would be expected from ether alone (Fig 31). Undoubtedly other influences of importance remain to be identified.

As originally introduced, the electroencephalogram afforded a crude index of cerebral cortical activity and one difficult of interpretation. Refinements have included analyses of the frequency of electrical tracings of certain cerebral cortical action potentials. Instruments have been designed to screen out certain frequencies and simplify the record.

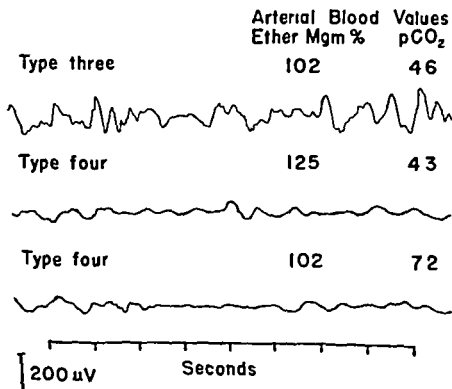


Fig 31 A comparison of the blood ether concentrations required to produce various electroencephalographic patterns in the presence of normal and elevated carbon dioxide tensions showing the greater degree of narcosis produced at a given ether level when arterial pCO_2 is elevated (After G H A Clowes, et al. *Ann. Surg.* vol 138 1953)

anesthesia so that the emergencies of incomplete anesthesia will not be encountered. It is easier to avoid trouble than to get out of it. A middle course, therefore, must be steered between a too light and an unnecessarily deep level of anesthesia.

A graphic presentation of the signs and stages of anesthesia is given in Figure 29.

THE ELECTROENCEPHALOGRAM

An interesting means of determining the depth of anesthesia has been the observation and analysis of the electrical activity of the cerebral cortex in man, as recorded in the electroencephalogram. Changes in electrical activity follow a characteristic pattern when most general anesthetics are given. Seven levels of brain depression from a waking pattern to a complete absence of electrical discharge have been described for ether (Fig. 30). Classification of patterns obtained in patients receiving cyclopropane and thiopental have also been made, and other anesthetics are being studied by this method.

The electroencephalogram is influenced in ways just now being understood. The addition of nitrous oxide to an ether anesthetic, for

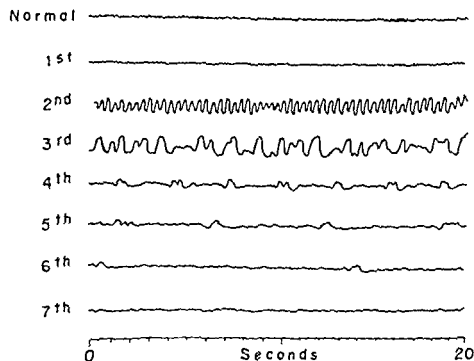


Fig. 30. Electroencephalographic patterns during increasing depth of ether anesthesia in man. Patterns 3, 4 and 5 occur during light, moderate and deep surgical anesthesia. Patterns 6 and 7 are seen during excessively deep anesthesia. (After A. Faulconer, Jr. *Anesthesiology*, vol. 13, 1952.)

example, influences the tracing. A lower concentration of ether is required to produce a given electroencephalographic level when nitrous oxide is present in the arterial blood in excess of 10 mg /100 ml. Carbon dioxide accumulation also affects the electrical pattern obtained from the brain. At a given arterial blood concentration of ether, the addition of carbon dioxide will produce an electroencephalographic level indicative of greater depression of the brain than would be expected from ether alone (Fig 31). Undoubtedly other influences of importance remain to be identified.

As originally introduced, the electroencephalogram afforded a crude index of cerebral cortical activity and one difficult of interpretation. Refinements have included analyses of the frequency of electrical tracings of certain cerebral cortical action potentials. Instruments have been designed to screen out certain frequencies and simplify the record.

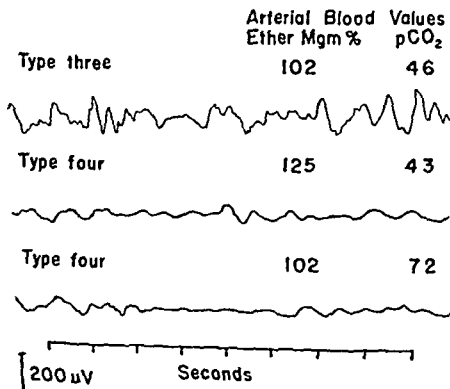


Fig 31 A comparison of the blood ether concentrations required to produce various electroencephalographic patterns in the presence of normal and elevated carbon dioxide tensions showing the greater degree of narcosis produced at a given ether level when arterial pCO₂ is elevated (After G H A Clowes, et al Ann. Surg. vol 138, 1953)

thereby. This has resulted in a more informative record. Nevertheless, the source of brain waves remains unknown, much of the interpretation is empiric and the practical utility of the record has not been established with certainty. It is our hope that research work in progress will permit greater usefulness of this objective measurement.

CONCENTRATION OF ANESTHETIC IN ARTERIAL BLOOD OR END-EXPIRATORY GAS

Another means for recording the depth of anesthesia is to measure the amount of anesthetic in arterial blood. A less satisfactory but more *simple variant is the measurement of gas concentrations at the end of expiration*, if an inhalation technique is being used. Gas sampling can be done via a catheter left in place in an endotracheal tube, through openings in devices which connect the breathing tubes of the anesthetic machine to the endotracheal tube, or through a needle placed in the expiratory breathing tube of a circle filter system close to the patient's face. The better the end-expiratory sample, the closer will this be to an alveolar sample. It is assumed that alveolar and arterial concentrations under normal circumstances are quite close.

If the concentration of an anesthetic at the end of expiration or in the arterial blood remains constant for 10 to 15 minutes, the vital organs—brain, heart, liver, kidney—will equilibrate with that concentration. A single sample cannot assure one that this reasonably steady state has been achieved, but if multiple samplings indicate this to be true, one can attempt a correlation between concentration of drug, the electroencephalogram or the clinical signs of anesthesia. Efforts in this direction reveal wide individual variability, reflected statistically in large standard deviations.

Pask and colleagues measured arterial blood concentrations in patients given morphine for preanesthetic medication and anesthetized with ether. A correlation was made between clinical estimation of the depth of anesthesia and blood levels obtained. Their data are listed in Table 9. The large standard deviations are evident.

Another instance of difficulty in correlation is the study of Faulconer and co-workers relating arterial blood levels of ether to electroencephalographic patterns. The subjects received pentobarbital and morphine prior to anesthesia. Data are given in Table 10. Large standard deviations are again obvious.

It becomes apparent that even with the brain at equilibrium with a given arterial blood concentration, the measurable and observable characteristics of patients differ considerably. Perhaps the course of

Table 9 Correlation of Clinical Signs and Ether Concentration

	STAGE III	NUMBER OF ANALYSES	MEAN ARTERIAL BLOOD ETHER (mg /100 ml)	STANDARD DEVIATION
Plane	1	43	48	14
Plane	2	37	74	16
Plane	3	58	105	21
Plane	4	10	135	22

Data from Hill, Matthews, Pask and Ritchie *Anaesthesia*, 7 243, 1952

Table 10 Correlation of EEG Level and Ether Concentration

ELECTROENCEPHALOGRAPHIC LEVEL	NUMBER OF OBSERVATIONS	MEAN ARTERIAL BLOOD ETHER (mg /100 ml)	STANDARD DEVIATION
1	11	52	24
2	22	60	26
3	48	84	21
4	107	103	23
5	37	120	27
6	2	147-198	—

Data from Faulconer *Anesthesiology*, 13 361, 1952

anesthesia in a given patient determines his response to a given arterial blood concentration at a particular time. If during a single anesthesia the patient has been subjected to a deep plane of narcosis once or twice with an inhalational anesthetic, the effects of this may not have been reversed or worn off as rapidly as one might believe despite the controllability afforded by the pulmonary route of administration. If this history of previous deep anesthesia has left an imprint on body cells and tissue function, a subsequent increase in concentration of anesthetic may cause a more marked response than if such an exposure had not occurred. This could explain the variability noted between arterial blood concentrations and a patient's response. We are not thinking of accumulation of anesthetic in the body, or of greater saturation of body tissues as time goes on. Rather there is the belief that reversibility of inhalational anesthetic effect may not be as rapid as has been supposed. If this be true, the effect of a given arterial blood concentration is a reflection in part of what has gone before during the same anesthetic.

Another difficulty with end-expiratory or arterial blood concentrations is noted when efforts are made to study the depth of anesthesia in

patients who are seriously ill. Smaller amounts of anesthetics are needed to produce comparable clinical levels of depression in "sick" patients as contrasted to those who are "fit." This matter is discussed on page 39.

SUMMARY

It must be emphasized that no single clinical sign can be used as a reliable indication of the depth of anesthesia. Signs vary from patient to patient, and from anesthetic to anesthetic. They are influenced by oxygen lack, carbon dioxide accumulation and unquestionably by other as yet unidentified factors. Even resort to objective methods is disappointing, for it is not always possible to correlate electroencephalographic tracings or blood concentrations of anesthetic with a clinical estimate of the degree of narcosis. Careful observation and minute to minute appraisal of all the clinical signs, together with whatever other objective measurements are available, provide the greatest safety for the patient.

REFERENCES

- Artusio, J. F., Jr. Di-ethyl ether analgesia. Detailed description of first stage of ether anesthesia in man. *J. Pharmacol. & Exper. Therap.* 11:343, 1954.
- Etsten, B., and Himwich, H. E. Stages and signs of pentothal anesthesia. *Physiologic basis Anesthesiology*, 7:536, 1946.
- Faulconer, A., Jr. Correlation of concentrations of ether in arterial blood with electroencephalographic patterns occurring during ether-oxygen and during nitrous oxide, oxygen and ether anesthesia of human surgical patients. *Anesthesiology* 13:361, 1952.
- Gillespie, N. A. The signs and reflex reactions of the stages of anesthesia. *Current Researches in Anesth. & Analg.* 22:275, 1943.
- Guedel, A. E. *Inhalation Anesthesia*. 2nd edition. The Macmillan Company, New York, 1951. pp. 10-52.
- Hill, F. W., Matthews, A. E. M., Pask, E. A. and Ritchie, L. W. Concentrations of di-ethyl ether in blood during surgical anaesthesia. *Anaesthesia* 7:243, 1952.
- Himwich, H. E. *Brain Metabolism and Cerebral Disorders*. The Williams and Wilkins Company, Baltimore, 1951.
- Laycock, J. D. Signs and stages of anaesthesia—a restatement. *Anaesthesia* 8:15, 1953.
- Mushin, W. W. The signs of anaesthesia. *Anaesthesia*, 3:154, 1948.
- Snow, John. *On Chloroform and Other Anaesthetics*. London, 1858, pp. 35-42 and pp. 87-97.
- Vandam, L. D. *Transport of Anesthetics and Stages of Anesthesia*. in Drill, V. A. *Pharmacology in Medicine*. McGraw Hill Book Company Inc. New York, 1954.

Fire and Explosion Hazard

IN COMPARISON with other hazards of anesthesia, explosions occur so infrequently as to be relatively insignificant. It is estimated that the mortality rate from explosions is about one in 1,500,000 anesthetics. The death rate directly attributable to anesthetics is difficult to determine, but in published reports has ranged from 1/350 to 1/4000. The emotional factors involved in an anesthetic explosion, however, make it feared out of proportion to its incidence.

Fires and explosions are combusive processes differing merely in the speed of reaction and the magnitude of forces released. Three elements are necessary for combustion: (1) a combustible substance, (2) a supporter of combustion, and, (3) a source of ignition.

All hydrocarbons are subject to decomposition by heat. Several burn readily or explode when in proper mixture with air, oxygen or nitrous oxide. The volatile liquids and gases used in anesthesia with the exception of nitrous oxide are hydrocarbons and may be classified as follows:

The vapors of divinyl and diethyl ether, ethyl chloride, ethylene and cyclopropane will explode under suitable conditions. Cyclopropane is alleged to detonate with greater violence. With the exception of ethylene the vapors of all these substances are heavier than air.

Trichlorethylene vapor is not flammable either in oxygen or air at ordinary temperature. Hydrochloric acid, free chlorine and traces of phosgene may be liberated if the vapor is heated.

Chloroform vapor will not ignite but will liberate phosgene if heated. Oxygen and nitrous oxide are not explosible, but support combustion. Explosions have occurred supposedly with nitrous oxide alone, presumably because of the presence of explosive contaminants derived from the anesthesia machine.

Nitrogen, helium and carbon dioxide, rather than being flammable, are flame-quenching substances

It is not necessary to memorize the flammable ranges for various anesthetics in air, nitrous oxide and oxygen. The following general knowledge should suffice

The ranges of flammability increase somewhat with nitrous oxide and markedly with oxygen. Thus, the general order of magnitude of the flammable range of anesthetics is 1.7 to 37 per cent in air, 1.4 to 40 per cent in nitrous oxide, 1.8 to 85 per cent in oxygen.

The concentrations of flammable agents required for anesthesia are within the explosive range.

Measures for the prevention of fires and explosions have been offered by engineers, fire underwriters and anesthetists. The most complete set of recommendations is that of the National Fire Protection Association. We endorse all protective measures which can be adopted within limits of practicability, unless their adoption involves substitution of a greater hazard. We do not believe that the final word has been written on this problem and that Ralph Waters' plea for the use of "applied brains" deserves as much consideration as does rigid adherence to measures upon some of which there is not yet general agreement. Our reasons for adopting this attitude include the following:

1. By rigid insistence on all N F P A recommendations, one may substitute a greater hazard for a lesser one. To illustrate—if a child is scheduled for fluoroscopically controlled reduction of a fracture under general anesthesia in a darkened room, the potential hazards of non-explosive thiopental or chloroform are greater than the risk of an explosion following use of ether.

2. Explosions have occurred in operating room suites of most modern design, in which all recommended preventive measures were ostensibly observed.

3. The evidence supporting certain claims is unconvincing, for example, the limits of resistance for conductive flooring set by the N F P A are not universally accepted.

4. There is a limit to the practicality of guarding against all explosive hazards. Conductivity of flooring cannot be guaranteed except at the time it was tested. If a floor is not properly cleaned, following a single day's usage its conductivity may change from an acceptable to a dangerous figure. Unless testing is carried out after each cleaning in each area—an impractical procedure—such a change may not be recognized. This reasoning also applies to the conductive rubber wheels of

anesthetic machines, operating tables and drag chains. The same sort of argument can be developed for other recommendations.

For reader interest and information we shall present in essence most of the recommendations of N F P A.

Storage of Agents and Care of Equipment

All cylinders, anesthetic machines with cylinders, and containers of volatile liquids should be stored in well ventilated places away from radiators, steam pipes and other sources of heat. Oxidizing substances (oxygen, nitrous oxide), supporters of combustion, should be separated from reducing substances (the hydrocarbons), the combustible substances. Under no circumstances should cylinders be refilled in the hospital. Oils and greases on equipment may be ignited by heat and static electricity generated by gases flowing under pressure.

Auto-oxidation of diethyl ether may form peroxides which are said to be violently explosive. Formation of such peroxides may occur when ether is allowed to stand in the vaporizer of an anesthesia machine not in use. Explosions which have originated within the anesthetic machine have been ascribed to this. For this reason, ether vaporizers should be emptied, wicks dried, and the machine "flushed out" with oxygen when not in use. Amber glass containers may help to prevent deterioration.

Removal of Anesthetic Vapors

Mechanical ventilation of anesthetizing locations is not a positive protection against anesthetic explosions. However, good ventilation is valuable in minimizing the possibility of explosion and is desirable for the comfort of personnel, removal of odors, and reduction of hazard from spillage of combustible anesthetics.

Closed System Technique

This system of administering anesthetics confines the explosive mixture to the apparatus and patient's respiratory tract. The hazard of ignition from exogenous sources is lessened, but a greater likelihood of serious injury to the patient results in the advent of an explosion.

Sources of Ignition Obvious sources of ignition such as flames, cigarettes and matches should be banned from anesthetizing locations (anesthesia rooms, operating theaters, corridors, recovery rooms and storage places).

Electrical wiring and equipment when installed in anesthetizing locations should conform to the specifications advised by the National

Electrical Codes and National Board of Fire Underwriters These specifications form the basis for local fire codes and regulations Among the suggestions are explosion proof switches, housings and outlets, grounded conduits, "kinkless" and rubber coated cords, spark-proof and enclosed motors

Static sparks probably constitute the greatest source of ignition They may be generated in many different ways, and are most difficult to control Everybody has had the experience of walking across a carpet and discharging a spark when contact was made with another person or object Whenever dissimilar materials are pressed or rubbed together electrical charges accumulate If the materials are conductors the charge will drain off but otherwise it will remain for long periods on the surface A spark will jump when charged objects are approximated Hazardous sparks can occur by induction at considerable distances from strong electrostatic charges

There can be no static sparking if all objects concerned are isoelectric, i e., at the same electrical potential This condition may be achieved by interconnecting all objects and persons within the "hazardous area" A list of suggested measures follows

Equipment In order for charges to be dissipated, the floor must provide a path of leakage from equipment and personnel Floors in anesthetizing locations should provide a path of moderate electrical conductivity for grounding The NFPA recommends that the measured resistance between electrodes three feet apart exceed 25,000 but be less than 1,000,000 ohms If these recommendations are followed, an ungrounded electrical system containing a ground warning indicator should be provided to protect personnel from electrical shock Ground is removed from an electrical system by use of an isolation transformer Floors must be kept clean of dirt and wax and should be tested at regular intervals for maintenance of conductivity by the hospital maintenance or engineering department

Furniture, equipment and operating tables should make contact with a conductive floor via metal or conductive rubber

Woolen or nylon blankets should be prohibited Cushions, mattresses and other interposed objects should be conductive The operating table should be fitted with a conductive strap to connect the bare skin of the patient to the ground Anesthetic machines should not be covered with dust draperies or plastered with adhesive tape because of the danger of static sparking when removed

High relative humidity (55 to 60 per cent) reduces the danger of static sparks In a closed system, rebreathed gases become completely

saturated with water vapor within a few minutes. Doubt has been cast on the practicality of deliberately wetting the reservoir bag and connecting rubber tubes before induction of anesthesia. Both interior and exterior of tubes and bag must be wetted to remove static charges. Greater protection is afforded by the use of conductive rubber. Maintenance of high humidity in the operating room is recommended.

Personnel Making or breaking connections on anesthetic apparatus should be carried out with both parts held by the anesthetist to maintain isoelectric conditions. If this is not done, sparking may occur between two pieces of metal at different electrical potentials.

Persons other than the anesthetist concerned should be kept away from areas of maximal accumulation of combustible substances. These are the patient's head and the anesthesia machine. If contact is necessary, it should be made at a distance from these danger points.

Movement of equipment and personnel, and handling of gases under pressure, should be done in a manner such as to avoid generation of static electricity.

All personnel in the anesthetizing areas should wear conductive footwear, that is, shoes with conductive soles or conductive "slip-ons." Ideally, footwear should be repeatedly tested to assure optimum conductivity (less than 1,000,000 ohms). Silk, nylon, rayon or sharkskin may be worn only beneath cotton outer garments. Nylon and silk stockings are permissible since there is close contact with the skin.

The Horton intercoupler which provides high resistance intercoupling (1 megohm) between persons, equipment and floor during operation is no longer a recommended safety device. The same results, that is, the maintenance of isoelectric conditions, can be achieved by applying the measures already outlined.

Cautery and High Frequency Equipment Surgical diathermy involves high frequency currents and cutting arcs or sparks at exposed electrodes. If the cautery is used during operations, it should be beyond a two foot radius from the head of the patient, and it should be used only if there is a suitable intervening barrier, and ventilation about the head. Most diathermy machines require a grounding electrode in contact with the patient. The current is led through this electrode and avoids sparking elsewhere. Occasionally the anesthetist may experience a shock when he touches the patient, or the patient may be burned at the grounding electrode or at contact with other metallic objects. This signifies improper grounding or a defect within the apparatus. A solution to the former is the use of wet cloths near the operative field for better grounding. In the latter case the machine must be repaired.

REFERENCES

- Beach, Robin Electrostatic Explosion Controls in Hospital Operating Rooms Transactions Paper No 56072, American Institute of Electrical Engineers 33 W 39th Street, New York 18, N Y
- Guest, P G , Sikora, V W , and Lewis, Bernard Static Electricity in Hospital Operating Suites Direct and Related Hazards and Pertinent Remedies Bureau of Mines Report of Investigations, No 4833, United States Department of Interior, January, 1952
- Recommended Safe Practice for Hospital Operating Rooms, 1956 Bulletin No 56 The National Fire Protection Association 60 Batterymarch Street, Boston 10, Massachusetts (The National Fire Protection Association standards of protection are purely advisory but are widely used by law-enforcing authorities in addition to their general use as guides to fire safety)

Arterial Hypotension during Anesthesia

THE MORE common causes of arterial hypotension encountered by anesthetists may be listed as follows

- 1 Excessive preanesthetic medication
- 2 The influence of potent therapeutic drugs used prior to anesthesia
- 3 Overdose of general anesthetics
- 4 Vascular absorption of local anesthetics
- 5 The circulatory effects of spinal and epidural anesthesia
- 6 Raised airway pressure
- 7 Hemorrhage
- 8 Surgical manipulation
- 9 Changes in position or motion of the patient
- 10 Abnormalities of the circulatory system

Each of these categories will be outlined briefly. Under each section prophylaxis will be considered. Treatment will be discussed at the conclusion of this chapter except for specific aspects which do not have general applicability.

Excessive Preanesthetic Medication

The tendency of opiates and opiate-like drugs to lower arterial blood pressure has been well documented. A number of pharmacologic actions contribute to this, including depression of the vasomotor center, reduction in skeletal muscle tone, depression of respiration, and dilation of peripheral blood vessels secondary to release of histamine or to a

direct action of the drugs. This circulatory depressant action often takes the form of postural hypotension, that is, an intolerance to the sitting or upright position in a patient who has received an opiate. Experimentally, passive tilt to the 50 or 60 degree head-up position can be used to bring out a latent derangement of the circulation induced by drugs (see p. 13). The assumption is that the opiates reduce the ability to compensate for circulatory stresses, such as hemorrhage, trauma or motion. The larger the dose of the opiate the more likely is this response. Rarely should one administer more than 10 mg. of morphine sulfate to healthy subjects prior to operation. In patients over 65 years of age opiates should be omitted altogether. A reduction in dosage is indicated in the debilitated or chronically ill.

The phenothiazine drugs, when used for preanesthetic medication, pose an even greater threat to the circulation than do the opiates. Chlorpromazine (Thorazine) is an offender, but promethazine (Phenergan) also has undesirable characteristics. The barbituric acid derivatives, such as secobarbital and pentobarbital, disturb the circulation least when given in 100 to 200 mg. doses prior to the induction of anesthesia.

It is sometimes difficult to judge accurately the amount of preanesthetic medication that will produce desired tranquillity without untoward side actions. It is obviously safer to err on the side of smaller doses than to seek marked sedation at the price of arterial hypotension or lessened ability to compensate for circulatory stress.

The Influence of Potent Therapeutic Drugs Used Prior to Anesthesia

Some of the drugs used in modern therapeutics can affect the course of anesthesia unfavorably. It is essential therefore that a careful history of all medication taken be part of the preanesthetic evaluation. Drugs of chief concern are the adrenal corticoids such as cortisone, and anti-hypertensive or tranquillizing drugs such as reserpine or chlorpromazine. Knowledge that a patient has been receiving these compounds may enable the anesthetist to avoid serious arterial hypotension.

Cortisone. Treatment with cortisone for five days or more is associated with distinct functional depression and histologic signs of atrophy of the adrenal cortex. In response to tissue injury, as with an operation, such a patient's adrenal cortex cannot produce a sufficient amount of corticoid to meet the demand. Hypotension is usually the chief manifestation of this adrenal insufficiency. Patients who have received cortisone must therefore be continued on the drug and receive increased amounts during anesthesia and operation.

The time required for return of human adrenal cortical function to normal after cessation of cortisone therapy is not known. Probably the intensity and duration of treatment are factors. If one is in doubt about adrenal cortical function, injection of ACTH and estimation of the urinary steroid excretion and the eosinophil response will indicate the extent of adrenal cortical suppression. In some clinics, patients with a history of having received cortisone within three months of a proposed operation will be given the drug again as a prophylactic measure. Other clinicians advise cortisone only if the patient has been given cortisone within three to four weeks. Regardless of this, if cortisone is used, doses are in the range of 100 mg. the day before operation, 200 mg. the day of operation, and decreasing amounts for the next three or four days if all goes well. On the day of operation hydrocortisone is given by slow intravenous infusion. When oral medication is tolerated this route is used postoperatively.

Reserpine Arterial hypotension has been reported following the administration of general anesthesia to hypertensive patients who have been receiving Rauwolfia or reserpine. Some workers believe that this untoward effect is related to increased vagal activity. The hypotension noted will frequently respond to a vagal blocking drug such as atropine given intravenously. How frequently this hypotension occurs is not known, nor has it been definitely related to dosage or duration of therapy. These drugs appear to exert a pharmacologic effect for 10 to 14 days after administration. If one wishes to be completely safe, and the patient's condition does not contraindicate withdrawal, treatment can be interrupted two weeks prior to a scheduled operation. In an emergency, use of a vagal blocking agent may be of value.

Chlorpromazine Arterial hypotension has been observed more frequently during anesthesia in patients receiving chlorpromazine. The duration of action of this compound is brief and operation can safely be undertaken within eight hours of interruption of therapy.

In all probability additional therapeutic agents will be developed which may pose a threat to patients during anesthesia and operation. The habit of asking about all medications is therefore essential. An anesthetist is likewise obligated to keep abreast of the possibilities of harm from new methods of treatment.

Overdose with General Anesthetics

Overdose with general anesthetics is one of the most common causes of arterial hypotension during anesthesia. Overdose may be "absolute" or "relative." In the former instance, an amount of drug is administered

in excess of that ordinarily tolerated by a normal patient. This may result from a sudden increase in the inspired concentration of an inhalation anesthetic, the intravenous injection of an excessive amount of a barbiturate, or the rapid absorption of a drug after rectal instillation. Acute overdosage is likely during the induction of general anesthesia.

Certain factors predispose to this type of "absolute" overdosage during anesthesia. As the patient's tissues approach saturation, the smaller is the increment of anesthetic required to cause a rise in blood stream concentration. Overdosage tends therefore to occur during prolonged anesthesia when large amounts of drug have been stored in the body. Overdose of irritant volatile liquids occurs more easily if the trachea has been intubated, for the protective action of laryngeal closure is no longer present and pulmonary uptake is great.

In the instance of "relative" overdose the actual amount of drug given is acceptable for the age and weight of the patient, but represents, at the time, more than he can withstand. This sensitivity may be more apparent than real. In the presence of a reduced circulating blood volume the concentration of an anesthetic agent increases more readily than in a larger volume. If vasoconstriction in other bodily areas occurs, and brain blood flow is maintained, excessive depression can result from the addition to the blood stream of ordinarily innocuous amounts of anesthetics. Under either circumstance a greater concentration of anesthetic would be presented to the vital organs than would be expected from the small amount of drug added (see p. 39).

The prevention of arterial hypotension due to overdosage involves two fundamental principles: (1) administration of the least amount of anesthetic compatible with adequate surgical working conditions, and (2) an agreement between surgeon and anesthetist that the optimal depth of anesthesia is that which is safest for the patient. For example, if marked abdominal muscular flaccidity is accompanied by hypotension, lesser relaxation must be maintained in the interest of increased safety.

Treatment of overdosage with inhalational agents consists of a prompt reduction in the anesthetic concentration inspired. If drugs are involved which must be destroyed within the body, or eliminated by routes other than the lungs, the general supportive measures listed at the end of this chapter are indicated until the offending substances have been removed.

Vascular Absorption of Local Anesthetic Agents

The rapid absorption of local anesthetics from mucous membranes or body tissues may cause marked hypotension (Chapter 16). The

probable causes for lowered blood pressure include depression of the myocardium, depression of the vasomotor center, and dilation of peripheral vessels resulting from a direct action. Prior administration of a barbiturate does not protect against toxic circulatory effects. An anesthetist should attend all critically ill patients scheduled for local anesthesia and those patients in whom extensive use of the method is planned regardless of physical condition. One of his responsibilities will be the prompt recognition and treatment of arterial hypotension.

This reaction can be minimized by reducing the total quantity of anesthetic drug injected per unit of time. Use of concentrated solutions and rapid injection are the chief factors responsible for lowered blood pressure (see Chapter 16 for proper dosage). When topical anesthesia is used the same principles apply. Application is made slowly and deliberately to avoid rapid vascular absorption. Cocaine (5 to 10 per cent), which limits its own absorption through a local vasoconstrictor action, is less likely to cause hypotension than tetracaine (Pontocaine) which is not a vasoconstrictor.

The treatment consists in use of the general supportive measures described at the end of this chapter.

The Circulatory Effects of Spinal and Epidural Anesthesia

One of the objections to the use of spinal anesthesia is the likelihood of a decrease in arterial blood pressure. In one series of 500 patients receiving spinal anesthesia, without prophylactic administration of a pressor drug, the systolic blood pressure declined 25 per cent or more from the preanesthetic level in 325 patients. The reasons for the fall in blood pressure have been discussed in Chapter 15. Briefly a reduction in total peripheral resistance and/or a decline in cardiac output have been observed. The former results from a paralysis of sympathetic vasoconstrictor fibers to arterioles. The latter follows pooling of blood in the postarteriolar blood vessels with a consequent reduction in venous return to the heart. The same alterations are associated with the arterial hypotension seen after epidural anesthesia.

A pressor substance, such as methoxamine (Vasoxyl), 10 to 20 mg, ephedrine 50 mg or phenylephrine (Neo-synephrine) 2 to 3 mg, injected intramuscularly five to ten minutes prior to administration of anesthesia will reduce the incidence of arterial hypotension considerably. These will not, however, prevent hypotension altogether. In our opinion this method of prophylaxis is indicated particularly in patients in the older age group in whom vascular reactivity may be lessened, and in all patients in whom a decrease in systemic pressure

may result in serious impairment of blood flow to such organs as the heart, brain or liver. In the latter group we would place patients with hypertension, coronary arterial disease, generalized arteriosclerosis, and those with a history indicating impairment of cerebral blood flow. If after prophylactic administration of pressor drug spinal anesthesia is not obtained, the rise in systolic arterial pressure resulting from the pressor drug rarely exceeds 40 mm Hg, an elevation that can occur in these patients from emotional upsets or exertion.

To prevent catastrophes associated with a sudden reduction in arterial blood pressure, the anesthetist must recognize that with spinal anesthesia such hypotension may develop almost immediately after injection of the local anesthetic into the subarachnoid space. Repeated measurements of blood pressure are therefore essential until the patient's reaction to the anesthetic is fully manifest.

Treatment must be prompt and energetic. Again it consists of those measures outlined at the end of this chapter.

Raised Airway Pressure

Positive pressure applied to the airway to ventilate the lungs may lower arterial pressure (Fig. 32). The raised airway pressure is transmitted to intrathoracic blood vessels. The greater the pressure, the less will be the blood flow. Cardiac output diminishes in proportion to the degree of interference with cardiac filling. As cardiac output and arterial pressure decrease, compensatory vasoconstriction is initiated via the sympathetic nervous system. Normal man can tolerate reasonable degrees of raised airway pressure through this ability to constrict peripheral vessels.

Certain changes occurring during anesthesia can either prevent or

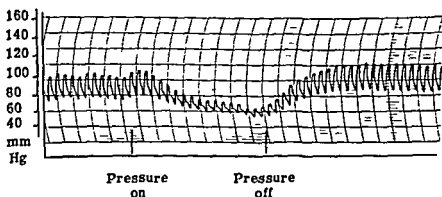


Fig. 32. Arterial hypotension following the application of 20 cm H_2O pressure to the airway of a patient under general anesthesia (Jones R. E. Helrich M. and Eckenhoff J. E. *Anesthesiology* vol 17 1956)

enhance the decrease in arterial pressure following raised pressure in the airway. These are

Venous Pressure Congested intrathoracic veins are less likely to collapse in response to external pressure than if venous pressure is normal. Cyclopropane and ether increase central venous pressure and afford a measure of protection. Similar protection can be noted in the presence of constrictive pericarditis, increased blood volume, or in congestive heart failure.

The Tone of Abdominal Muscles If the abdominal wall resists the transmitted intrapulmonic pressure, greater pressures are transmitted to intrathoracic structures and more hypotension results. Increasing depth of general anesthesia, spinal anesthesia, or the use of muscle relaxants may afford protection against raised airway pressure through the production of abdominal muscular relaxation.

Peripheral Vasoconstriction Ganglionic blocking agents, spinal or epidural anesthesia, the release of histamine following the use of opiates or *d*-tubocurarine, and the vasodilator effect of general anesthetic agents, all may decrease the ability of the patient to constrict blood vessels. Compensatory constriction is less adequate or absent under these circumstances and raised airway pressure consequently produces greater degrees of arterial hypotension.

The prevention and treatment of hypotension consists in decreasing the amount and duration of the pressure applied to the airway. If at the same time pulmonary ventilation becomes inadequate, intermittent positive-negative respiration provided by mechanical ventilators may be useful, or a return to spontaneous breathing may be permitted.

Hemorrhage

Arterial hypotension may follow blood loss. Clinical estimation of the amount of blood lost during an operation is often faulty. The technique of weighing sponges and subtracting the dry from the wet weight permits a more accurate appraisal of the loss. Replacement of blood loss rests then on a sounder basis.

The diagnosis of hidden hemorrhage is not always easy. In the immediate postoperative period occult intrathoracic or intra-abdominal bleeding can be the basis for progressive hypotension. If the patient lies supine, tachycardia is often absent until blood loss is considerable. Bradycardia is frequently seen. A pale, moist skin and a narrowed arterial pulse pressure are suggestive.

Hemodilution takes place relatively slowly, so that determination of hemoglobin or hematocrit values is not of great help. Determinations

of blood volume are not helpful unless preoperative control readings have been made or unless the degree of blood loss exceeds the maximal error of current blood volume methods, or 30 per cent of the original value. It has been suggested that the head up tilt test be used, searching for tachycardia and hypotension, but this is a non-specific circulatory response and is not pathognomonic of hemorrhage. Occasionally in women collection of intraperitoneal blood can be demonstrated by vaginal examination. It is evident that clinical judgment must be at its keenest in the diagnosis of hemorrhage.

If blood loss of major degree is anticipated during operation, a large bore needle (15 gauge) should be placed into a vein so that blood can be administered rapidly. Blood may have to be given under pressure. If pressure is applied to the surface of blood within a container, the danger of air embolus is great unless the utmost vigilance is exercised.

The value of having sufficient whole blood, typed and crossmatched, stored in the operating room refrigerator prior to elective major operations is accepted by most workers. In emergencies, type O universal donor blood of low A and B titer can be used. Two or three units can be alternated with a unit of Dextran.

It must be recalled that the following hazards may be associated with blood transfusion: hemolytic reactions, transmission of infectious hepatitis, pyrogenic reactions, sensitization to subsequent transfusions, transfusions of massively contaminated blood, and, in chronically anemic patients and patients with congestive heart failure, overloading of the circulation and pulmonary edema. Blood should therefore be administered only when absolutely indicated. A reasonable rule of thumb is that if 500 ml. of whole blood is all that is to be given to replace loss in adults, it is probably wise to omit blood altogether and use the same volume of a plasma expander such as 6 per cent Dextran. The patient can add hemoglobin and red blood cells to his circulation in the normal manner during convalescence and may have been spared one or more of the dangers of transfusion.

Surgical Manipulation

Another common cause of arterial hypotension is that related to surgical manipulation. Hypotensive episodes are explained usually on a mechanical or reflex basis. In the former, venous return to the heart may be obstructed by packs, through torsion or compression of large veins by retractors, by compression of gallbladder or kidney rests, or by the weight of the gravid uterus or large abdominal tumors as the patient lies supine. The sudden reduction of a previously increased

intra-abdominal pressure, during rapid drainage of massive ascites or the delivery of a large tumor mass, may be followed by arterial hypotension. This may be due to pooling of blood in dilated veins.

Reflex hypotension may follow traction on such structures as the gallbladder (Fig 33), appendix (Fig 34), uterus or mesentery (Fig 35), or mere stimulation of the parietal peritoneum in the upper abdomen. If the patient is conscious at the time, such manipulations may also cause pain, nausea, vomiting and shortness of breath. Reflex hypotension is not limited to stimulation of intraperitoneal tissues. It may also follow manipulations in the chest or those involving the periosteum or joint cavities. It is presumed that the autonomic nervous system is involved in these reactions, but whether the afferent impulses of this "traction reflex" ascend via sympathetic or vagal pathways is not clear. In the upper abdomen the intercostal nerves carry many of these impulses. The efferent path is probably parasympathetic with the cardiac vagus playing a major role. The effects of depth of anesthesia and concurrent use of muscle relaxants on the incidence and severity of traction should be mentioned. Some evidence exists that reflexes are more active during light planes of general anesthesia and may be partially or completely blocked in deeper planes, or by the use of those relaxants which interfere with ganglionic transmission (see

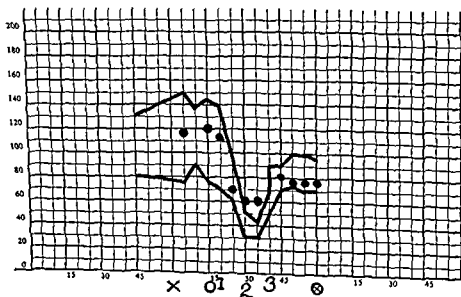


Fig 33 Hypotension in a 78 year old patient undergoing a cholecystostomy. One per cent procaine used for regional anesthesia by field block. 1 Peritoneal cavity opened. 2 Patient cold and clammy retching. 3 Bed of gallbladder infiltrated with procaine. Solid lines are systolic and diastolic blood pressure, dots represent pulse rate.

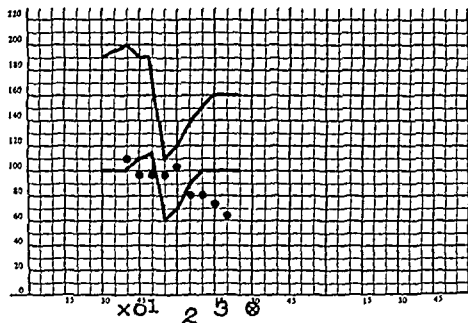


Fig 34 Hypotension in a 45 year old patient with hypertension and hyperparathyroidism undergoing an appendectomy One per cent procaine field block anesthesia 1, Traction on appendix—pain 2 Moaning—feels faint 3, 'Better now'—traction off

Chapter 12) Minimal general anesthesia, essentially analgesia, may also be associated with lessened reflex responses These aspects deserve further study

Gentleness on the part of the surgeon and awareness of the consequences of certain surgical manipulations constitute the basis for the prevention and treatment of this type of arterial hypotension

If hypotension is accompanied by bradycardia, that is, a heart rate of 60 or less, the intravenous injection of 0.3 to 0.4 mg atropine sulfate may return the blood pressure towards more normal levels Ephedrine (15 to 25 mg intravenously) is often effective under these circumstances Since this drug acts primarily to increase myocardial con-

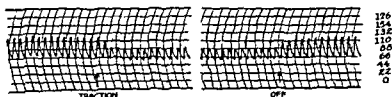


Fig 35 Continuous intra arterial pressure tracing showing abrupt reduction of pulse pressure with traction on the mesentery of the colon of an anesthetized patient Numbers at right are mm Hg (Eather, K F Peterson, L H and Dripps, R. D Anesthesiology, vol 10 1949)

tractility, its ability to reverse hypotension associated with traction supports the belief that the lowered blood pressure may be related to a reflexly induced (vagal) reduction of cardiac force

Changes in Position or Motion of the Patient

The circulatory system of the anesthetized patient is less able to compensate for stress than that of the unanesthetized. This is particularly true of critically ill individuals. Movement of these patients, as for placement in the lateral decubitus, may cause a marked reduction in arterial pressure. All changes of position should be accomplished slowly and gently, and blood pressure levels must be determined immediately after the change in position. Prompt treatment then can be instituted if necessary.

Certain positions needed for a particular operation may be poorly tolerated. The lateral flexion required for exposure of the kidney or adrenal gland is one example. Operations performed in the sitting position are not infrequently complicated by postural hypotension. In patients in poor physical condition it is wise to test the cardiovascular response to the position needed before anesthesia is induced.

This relationship of position and motion to the onset of lowered blood pressure is discussed in greater detail in Chapter 23.

Abnormalities of the Circulatory System

Myocardial infarction during anesthesia may be followed by a profound fall in blood pressure. The diagnosis of this complication is difficult to make and is rarely established until the ischemia is of some duration. Hypotension may also accompany paroxysmal tachycardia such as occurs in patients with mitral stenosis who have not been adequately digitalized. Multifocal ventricular tachycardia, noted not uncommonly during cyclopropane anesthesia, may be so rapid as to compromise diastolic filling of the heart. Blood pressure then declines. Thyrotoxicosis may rarely be associated with a tachycardia of sufficient rate to cause hypotension.

Another cause of lowered arterial blood pressure, difficult to diagnose, is embolism to any of several vascular beds. Embolism to the brain is a precipitating factor in hypotension sometimes seen during mitral commissurotomy. Pulmonary emboli from peripheral veins may occur, as may fat emboli from fracture sites, or amniotic fluid emboli during delivery.

Hypovolemia may be present prior to operation in certain patients. Dehydration, hemorrhage, loss of gastrointestinal fluids, and burns

may contribute to this state. If these remain unrecognized and untreated, the dilatation of the vascular bed associated with many types of anesthesia can be followed by arterial hypotension. The volume of other fluid compartments may also be a factor in circulatory adequacy. These are inextricably related to electrolyte concentrations, so that one must recognize that both fluid and electrolyte imbalance can be associated with hypotension.

A hitherto unrecognized irritable carotid sinus can be stimulated by local manipulation occurring during anesthesia. Pressure in the neck may give rise to hypotension on this basis.

TREATMENT IN GENERAL

General supportive measures for the circulation that can be adopted during anesthesia include, mainly, the parenteral administration of fluids and the injection of pressor drugs.

Fluid Therapy

The rapid intravenous administration of one of a variety of solutions may be life-saving during severe arterial hypotension. The volume of fluid given rather than its composition is vital during the early moments. The initial selection is therefore based on what is immediately at hand. Physiological saline, 5 per cent glucose in water, a plasma expander such as 6 per cent Dextran, one of the gelatins, or human serum albumin can be given rapidly until whole blood becomes available.

Pressor Drugs

There are two broad groups of pressor substances: (1) those with an action directed primarily toward increasing arteriolar and venous tone, e.g., methoxamine (Vasoxyl), phenylephrine (Neo-synephrine) and norepinephrine (Levophed) and (2) those which primarily increase the strength of cardiac contraction, e.g., ephedrine and epinephrine. This classification is only relatively accurate since most of these drugs act to increase both vascular tone and myocardial contractility. It is the greater effect in one area that justifies the classification.

The first approach to hypotension should consist of an initial intravenous and intramuscular injection of a pressor drug. If this is ineffective or if the effect is evanescent, one should resort to continuous intravenous infusions for a more consistent and prolonged effect. Occasionally the pressor drugs may be ineffective. Loss of vascular reactivity to these compounds has been described by certain workers, who allege that the intravenous administration of 100 to 200 mg. of

hydrocortisone will restore this reactivity. The data supporting this contention are not yet convincing, but the use of hydrocortisone in this dosage is justified in the management of intractable hypotension.

The suggested dosage range for adults of a few of the commonly used pressor drugs is as follows:

	SINGLE INJECTION I M (mg)	SINGLE INJECTION I V (mg)	CONCENTRATION FOR CONTINUOUS I V INFUSION
Methoxamine	5-20	5	—
Phenylephrine	2-3	0.2-0.5	10 mg /500 ml
Norepinephrine	—	—	4 mg /500 ml.
Ephedrine	25-50	5-15	—
Epinephrine	—	0.1-0.5	—

Unusual Complications of Anesthesia

THE ADMINISTRATION of anesthesia involves the avoidance and treatment of many complications. These may be inherent in the patient, in the agents employed, in the technique of anesthesia, or in the supportive measures used. Some of these complications have been discussed in other sections. Those listed here occur with sufficient frequency and severity to warrant separate mention and emphasis.

INJURIES TO THE EYES

Careless application of a face mask, certain positions of the patient on the operating table, the anesthetic technique employed, or the activities of the surgeon in preparing the skin and operating on the head or neck may predispose the patient to ocular injury. Open techniques with volatile liquids are particularly likely to lead to conjunctivitis or corneal abrasion. Large masks pressing on the orbits have produced intraocular injuries, periorbital edema, postoperative numbness in the area of the supraorbital nerves, and reflex vagal responses as evidenced by cardiac slowing.

In general, the best precaution against corneal injury is to keep the eyelids closed. We do not advise instillation of liquid paraffin into the conjunctival sacs because anesthetics soluble in oil may accumulate in the paraffin. The practice of eliciting a corneal reflex to determine depth of anesthesia is not a good one because of the possibility of injury to the cornea. It may be necessary to approximate the lids with cellophane tape or adhesive to keep the eyes closed, particularly during long procedures. Instillation of 5 per cent boric acid ophthalmic oint-

ment or "artificial tears" now made by several manufacturers will prevent drying of the cornea in deeper planes of anesthesia

The tendency to call all ulcerations of the cornea or conjunctiva following open-drop anesthesia "ether ulceration" is incorrect. Ether dropped directly into the eye will produce epithelial ulceration that will heal normally within 24 hours. Ulceration is more likely to result from trauma to the eye or drying of the eye due to carelessness on the part of the anesthetist.

The eyes should be inspected at the conclusion of anesthesia. If injury has occurred, *immediate* ophthalmologic consultation must be sought. Simple conjunctivitis is best treated by irrigations with saturated solutions of boric acid. Corneal abrasions are not only painful but may progress to inflammation of the uveal tract. If treated early with antibiotics instilled locally, abrasions will usually reepithelialize within 24 hours. Pupillary dilators may be used to prevent synechiae when inflammation is present.

INJURIES TO THE LUNGS

Excessive Pressures

Since the gases used in anesthesia are stored at high pressures, the possibility of pressure damage to the lungs is ever present. These injuries can occur not only during anesthesia but whenever inhalational therapy or resuscitation is applied. The exact limits of pressure that can be safely applied to the lungs have never been clearly defined and differ from patient to patient. It is said that the pressures should not exceed 20 to 30 cm H_2O , yet greater pressures may be needed to inflate the lungs. Whether damage will occur depends on the rapidity of development, or the distribution of the pressure, and whether the thorax is open at the time.

If high pressures are transmitted to the alveoli, rupture may take place with subsequent hemorrhage and capillary air embolism, or dissection into the interstitial tissues (Fig. 36). Air in the interstitial tissues of the lungs may rupture into the pleural cavities or dissect back through the hilum into the mediastinum (pneumomediastinum). The lung may remain inflated with air (pulmonary interstitial emphysema). Venous return to the heart may be impeded by air in the mediastinum (air block). Mediastinal emphysema and air block can be recognized by distinctly audible churning sounds synchronous with the heart beat (Hamman's sign). Air in the mediastinum can dissect further retroperitoneally or subcutaneously, or may escape into the pleural cavities when the mediastinal pleura ruptures.

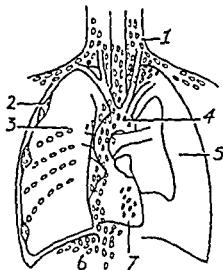


Fig 36 Escape of air following rupture of the lungs 1 Subcutaneous emphysema 2 Subpleural bleb 3 Pulmonary interstitial emphysema 4 Mediastinal emphysema 5 Pneumothorax 6 Pneumoperitoneum 7 Air embolus to the heart

These accidents are especially prone to occur when there is free access of pressure to the alveoli, as when the glottis is held patent with an endotracheal tube. Pneumothorax, mediastinal and pulmonary interstitial emphysema, and capillary air embolism must be recognized and treated at once if the patient is to survive. Often there may be little one can do, but the following measures are suggested.

For pneumothorax—aspiration of pleural air and institution of underwater negative pressure drainage. At the same time pressure should be applied to the upper airway to assist in reexpanding the lungs.

For mediastinal and subcutaneous emphysema—a superior mediastinotomy may be performed under local anesthesia if the gas is under pressure and causing respiratory or circulatory embarrassment.

For air embolism—the patient should be turned head downward in a steep Trendelenburg position with the right side uppermost to forestall air tamponade at the outflow tract of the right ventricle. If air is heard within the heart, an attempt can be made to aspirate it. If the heart action stops, cardiac resuscitation as described in Chapter 27 should be undertaken. Manual systole of the heart may help the passage of air emboli lodged in the coronary arteries. The head-down position may prevent cerebral and coronary air embolization.

Protection against these accidents can only be provided by the anesthetist's careful actions and the obligatory placement of safety valves between the source of pressure and the patient's airway. As a general

rule gas cylinders should be opened and, where possible, flows adjusted before connection is made to the patient

Insufflation of Liquid Ether into the Lungs

The design of some anesthetic apparatus with respect to vaporization of volatile liquids allows the possibility of insufflation of liquid ether to the lungs. The likelihood of this accident is increased during endotracheal anesthesia. Liquid ether will produce an immediate intense bronchoconstriction and subsequent pulmonary edema. Precautions to avoid this catastrophe include the placement of a trap between the vaporizer and the patient, adjusting gas flows with the vaporizer out of the circuit, and making these adjustments before connecting the apparatus to the patient. Immediate treatment should consist of oxygenation of the lungs and lavage of the trachea with sterile saline solution. Should the victim survive, inflammatory reaction and pneumonia will follow.

INJURIES TO THE NERVES

Nerves can be damaged during anesthesia through stretch or compression. This occurs because the anesthetized patient cannot perceive pain and lacks protective muscle tone. Among the nerves commonly injured are those of the brachial plexus, the ulnar or radial nerve, the common peroneal nerve and the facial nerve.

The nerves comprising the brachial plexus are fixed centrally at the transverse processes of the vertebrae and peripherally at the attachment to the arm. Separation of these two points may stretch the nerves with resultant molecular damage, hemorrhage or ischemia and development of palsy (Fig. 37). Flexion of the head to one side with coincident downward or backward displacement of the shoulder can place tension on the nerves. The presence of several natural anatomic fulcrums such as the scalene muscles, the attachment of the pectoralis minor muscle to the coracoid process of the scapula, and the rounded head of the humerus provides additional possibilities for tension. Hyperabduction of the arm stretches the brachial plexus around several of these fulcrums. The use of a shoulder brace, improperly applied to the soft tissues of the cervical triangle, may not only act as an artificial fulcrum but also compress the brachial plexus against underlying structures. A shoulder brace should not be used when the arm is already extended on an arm rest. Lastly, the plexus may be pinched between muscles and bone, or between the clavicle and first rib.

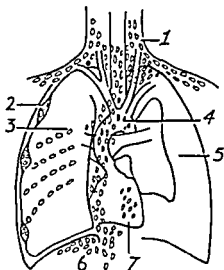


Fig 36 Escape of air following rupture of the lungs 1 Subcutaneous emphysema 2 Subpleural bleb 3 Pulmonary interstitial emphysema 4 Mediastinal emphysema 5 Pneumothorax 6 Pneumoperitoneum 7 Air embolus to the heart.

These accidents are especially prone to occur when there is free access of pressure to the alveoli, as when the glottis is held patent with an endotracheal tube. Pneumothorax, mediastinal and pulmonary interstitial emphysema, and capillary air embolism must be recognized and treated at once if the patient is to survive. Often there may be little one can do, but the following measures are suggested.

For pneumothorax—aspiration of pleural air and institution of underwater negative pressure drainage. At the same time pressure should be applied to the upper airway to assist in reexpanding the lungs.

For mediastinal and subcutaneous emphysema—a superior mediastinotomy may be performed under local anesthesia if the gas is under pressure and causing respiratory or circulatory embarrassment.

For air embolism—the patient should be turned head downward in a steep Trendelenburg position with the right side uppermost to forestall air tamponade at the outflow tract of the right ventricle. If air is heard within the heart, an attempt can be made to aspirate it. If the heart action stops, cardiac resuscitation as described in Chapter 27 should be undertaken. Manual systole of the heart may help the passage of air emboli lodged in the coronary arteries. The head-down position may prevent cerebral and coronary air embolization.

Protection against these accidents can only be provided by the anesthetist's careful actions and the obligatory placement of safety valves between the source of pressure and the patient's airway. As a general

INJURIES ASSOCIATED WITH INTRAVENOUS ANESTHESIA AND THERAPY**Traumatic Venipuncture**

An improperly performed venipuncture can cause great discomfort to the patient through pain or in subsequent hematoma formation. We urge that local anesthesia be employed for the insertion of large gauge needles and advise against multiple attempts at venipuncture when the puncture is not an essential part of the anesthetic procedure.

Extravasation of Intravenous Barbiturates

Because of the intense alkalinity, extravasation of strong solutions of intravenous barbiturates (2.5 to 5 per cent) can lead to tissue necrosis and indolent ulcerations. Neuritis has been noted to follow extravasation about a nerve. If blood flow in a vein is sluggish or obstructed, pooling of barbiturate may be followed by thrombophlebitis. When extravasation or pooling occurs the alkaline solution can be diluted and neutralized by the injection of 1 per cent procaine hydrochloride. At the same time pain and vasospasm may be relieved.

Accidental Intra-arterial Injection of Intravenous Barbiturates

A rare but disastrous accident is the inadvertent injection of thiopental or thiamylal into an artery. This can occur more easily in the antecubital space where the brachial artery and antecubital veins are in close proximity. When, after injection, the onset of anesthesia is delayed, if the blood is bright in color or ejects the barrel of the syringe, one should suspect intra-arterial injection. The patient will experience intense pain as if scalding water were poured over the extremity. Vasospasm, blanching and eventually ischemic necrosis of tissue will follow. Hands and arms have been lost from gangrene. The treatment is discussed in Chapter 14.

Miscellaneous Accidents

There are an infinite number of rare accidents that may happen during or following anesthesia. The following is a partial list of such complications observed during the past two decades.

- 1 A child unattended has fallen from an operating table and sustained multiple lacerations.
- 2 A man carelessly positioned for a lumbodorsal sympathectomy has awakened with pressure contusions of the genitalia.
- 3 A neurosurgical patient has sustained third degree burns of the

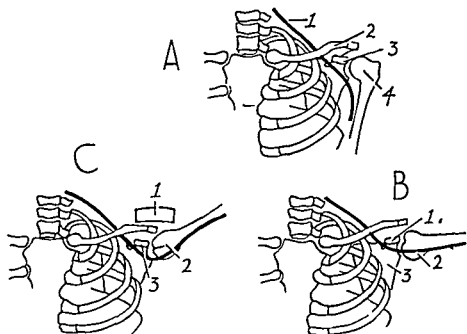


Fig 37 Traction on the brachial plexus A Arm at the side 1 Brachial plexus 2 Clavicle 3 Coracoid process of scapula 4 Head of humerus B Arm at right angle Scapula (3) begins to rotate and brachial plexus is stretched only slightly beneath the coracoid process (1) and around the head of the humerus (2) C Arm hyperextended with the shoulder brace (1) depressing the scapula Brachial plexus stretched beneath the coracoid process and around the head of the humerus

To avoid brachial plexus injuries one must bear the possibility constantly in mind and avoid extremes of position of the head and arm. When palsy results after operation, a careful neurologic examination must be made and immediate measures for restoration of function begun. The latter include proper support of the arm and physiotherapy. In the case of severe injuries, restitution of normal function may not take place until six months or a year has elapsed.

The ulnar and common peroneal nerves and to a lesser degree the radial nerve are superficial structures that may be compressed against bone or stretched around bony eminences. Certain operating positions such as the lithotomy or lateral decubitus will predispose these nerves to injury. If the latter occurs, treatment is the same as that described for brachial plexus palsies.

The facial nerve may be injured by overenthusiastic attempts at elevating the jaw by pressure on the rami of the mandibles. Weakness of the muscles around the mouth is a manifestation of this injury. It is usually reversible within one to two weeks.

Surgeon-Anesthetist Relationships

SURGEONS and anesthetists comprise a team of physicians dedicated to the welfare of the surgical patient. The patient's interests are best served if each member of the team recognizes his responsibilities and is at the same time aware of the problems faced by his colleagues. So far as the anesthetist is concerned, his position on the team is clearly defined. He must be thoroughly acquainted with the patient's medical history, the operation proposed, and the possible risks involved. He must understand the stresses that will be placed upon the patient's bodily systems by the contemplated anesthesia and surgical procedure. He must confer with the surgeon and other consultants concerning aspects of the patient's disease with which he is unfamiliar. He must keep the surgeon informed of the patient's progress, not by the routine recitation of vital signs but by the instant transmission of important information. He must constantly monitor a multitude of details that would distract the surgeon's attention from the technical problems of the operation. He must closely supervise the patient's care in the crucial immediate postoperative period when the surgeon is elsewhere.

The surgeon should consult with the anesthetist before operation and discuss the details of surgical management. He must not insist upon unnecessary speed in induction of anesthesia since this can be detrimental to the patient. He should rarely demand a particular anesthetic or technique since he may not know the limits of the anesthetist's capabilities or the potentialities of the anesthetic agents and techniques. He must not expect cadaveric relaxation if this be not needed or if it cannot be produced with safety. He must understand the problems

legs from an exposed electric light bulb used to illuminate the area under the operating table

4 A patient has developed marked swelling resembling parotitis because of manual efforts to elevate the mandible during respiratory obstruction

5 A patient's false teeth removed just prior to anesthesia and care fully wrapped in gauze have then been carelessly thrown away

6 A young boy for tonsillectomy has struggled during induction, struck his head against the edge of the table, and required suture for a laceration of the scalp

7 A pint of blood has been allowed to extravasate into the tissues of the arm positioned out of sight under the surgical drapes

It can be seen that most of these complications could and should have been avoided

REFERENCES

- Gillespie N A Surgeon anesthetist relationships J A M A , 118 787, 1952
- Griffith, H R Position of the anesthesiologist on the hospital staff Current Researches in Anesth & Analg , 29 189 1950
- Lortie, D C The sociologist looks at the profession of anesthesiology Current Researches in Anesth & Analg , 29 181, 1950
- Orton, R H The surgeon anesthetist relationship M J of Australia, p 239, August 13, 1949
- Wiley v Wharton 41 NE (2d) 255 (Ohio, 1951)
- Woodson v Huey 2 CCH Neg Cases 2d 284 Oklahoma Supreme Court, June 23 1953

peculiar to anesthesia and be prepared to allow time for the anesthetist to solve them. He must upon occasion agree to a rapid conclusion of the operation if the anesthetist believes that the patient's condition is deteriorating.

A team is at its best when its members have worked together repeatedly. It takes time for an individual to learn where he fits into the scheme of things and how his associates perform their particular tasks. Good teamwork comes out of mutual respect. A new member of the team must prove himself worthy before he is accepted upon the same basis as the others.

Surgical teams work under a variety of circumstances. In most operations there is little reason for tension, but in difficult operations or those in which unexpected complications arise, tempers may flare and harsh words may be exchanged. The care of the patient may suffer thereby. It is important for surgeon and anesthetist each to *visualize the other's predicament*. Often technically simple operations may have to be performed under the most trying anesthetic conditions. On the other hand, an intricate and difficult surgical procedure can be performed without anesthetic incident. If irritability is displayed by either member of the team, the other must assess the situation and minimize friction if possible. The operating room is not the place for verbal battle. Words spoken in anger can be withdrawn at the conclusion of operation but if the patient has suffered, irreparable damage may ensue.

The inexperienced anesthesiologist may sometimes fear that he will occupy a position subordinate to that of the surgeon. This is a fear arising from lack of actual experience in the team performance of intricate surgery, although the anesthesiologist may himself contribute to his subordinate status by failing to participate fully in the medical care of the patient. The evidence that he is first a physician and then a *competent anesthetist* will assure his acceptance as an equal with the surgeon in the over-all care of the patient.

Confusion often exists in the minds of surgeons and anesthetists alike as to the medico-legal responsibility in anesthesia. A study of court rulings indicates that a surgeon is responsible for the anesthetic only if it is administered by a technician under his direction. If the anesthetic is given by an anesthesiologist, he is legally responsible for the choice of agent and the care of the anesthetized patient.

One might summarize the essentials of good surgeon-anesthetist relations as being mutual professional confidence, understanding, frankness, honesty, courtesy and fair-mindedness.

REFERENCES

- Gillespie, N A Surgeon anesthetist relationships J A M A , 118 787, 1952
Griffith H R Position of the anesthesiologist on the hospital staff Current Researches in Anesth & Analg , 29 189 1950
Lortie, D C The sociologist looks at the profession of anesthesiology Current Researches in Anesth & Analg , 29 181, 1950
Orton, R H The surgeon anesthetist relationship M J of Australia p 239, August 13, 1949
Wiley v Wharton 41 NE (2d) 255 (Ohio, 1951)
Woodson v Huey 2 CCH Neg Cases 2d, 284 Oklahoma Supreme Court, June 23, 1953

IV

The Postoperative Period

Hazards of the Immediate Postoperative Period

AS AN operation draws to a close there is a natural tendency for all concerned to "let down." The pressures associated with the surgical procedure are over, and with the understandable desire to relax, attention may be diverted from the patient. The surgeons usually leave the room. Often the anesthetist is left alone, yet at this time a number of events transpire which pose a threat to the patient's welfare. Removal of an endotracheal tube can be followed by many hazards. If a high oxygen concentration has been used during anesthesia, its replacement with room air can have untoward consequences. Vomiting may occur. Emergence excitement may develop. Pain may begin to be experienced by the patient, and fear and anxiety become manifest. The patient's position is changed as he is moved from operating table to bed or litter. Atelectasis, emboli, pneumothorax or the sequelae of inadequate treatment of blood loss may escape detection. The consequences of these and other problems will be discussed.

ARTERIAL HYPOTENSION

When the anesthetist is busy disconnecting the anesthetic machine, turning off cylinders and making preparations to move the patient from the operating room, he may not observe blood pressure or pulse for a five to fifteen minute interval. A decrease in blood pressure is not uncommon at this time, and may have reached serious proportions before it is recognized. The following factors have been identified as contributing to this inadequacy of the circulation, probably others exist.

Change of the Patient's Position or Motion during Transportation

It is known that passive tilt to the head-up position can be followed by hypotension even in normal subjects. If compensatory reflexes are impaired by narcotics, ganglionic blocking drugs or local vasodilator substances, postural hypotension may be marked (see p 155). The mechanism appears to be pooling of blood in the dependent lower extremities with a reduction in circulating blood volume. This alteration is probably the basis for the hypotension that develops when the patient's legs are returned from the lithotomy to the supine position at the end of operation (Fig 38). Awareness of this possibility is essential. Prophylaxis consists in binding the legs from toes to groin with tight bandages prior to change in position. This precaution is also indicated at the end of prolonged operations, after those accompanied by major degrees of blood loss, and in debilitated patients. Despite this precaution, blood pressure should be recorded as soon as the legs are lowered. If hypotension occurs, the legs should be elevated again. Change in position from the lateral to the supine may also be followed by hypotension.

Movement of the anesthetized patient can cause a reduction in blood pressure. This may follow transfer from table to litter or bed, wheeling

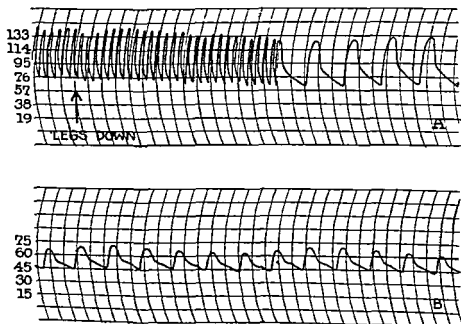


Fig 38 Postural hypotension developing after patient who had been in the lithotomy position was placed supine. Time between A and B in the arterial pressure tracing was two minutes. Numbers represent mm Hg pressure.

the litter from the operating room, or, if the patient is taken directly to his room, the jarring motion of an elevator. We have seen cardiac arrest as a patient was lifted from the operating table and transferred to the litter. Similar catastrophes have occurred during transport of anesthetized patients in elevators. The physiologic basis for this hypotension has not been determined, but pooling of blood in the periphery, perhaps in the deep back muscles, is probable. Stimulation of cutaneous or proprioceptive receptors by jouncing may be involved, since hypotension has been observed as air currents were blown over the skin of anesthetized patients, or as a painless skin incision was made in a patient anesthetized by subarachnoid block. Local axon reflexes leading to redistribution of blood may be the factor here. The hypotension associated with change of position and movement demands further study.

Reduction in Level of General Anesthesia

As an inhalational drug is eliminated, non-volatile depressants metabolized, or muscle relaxants detoxified, changes in the circulation take place. "Cyclopropane shock" is a case in point. At the end of an anesthesia with this substance, hypotension may develop. Reduction of a previously elevated arterial carbon dioxide tension has been implicated. The thesis is that inadequate pulmonary ventilation during anesthesia occurs more often than is appreciated. As a result, carbon dioxide accumulates and tends to elevate blood pressure through centrally mediated vasoconstriction. As anesthesia terminates and alveolar ventilation improves, arterial carbon dioxide tension is lowered. With a lessened central stimulation vasomotor activity decreases and blood pressure falls. A similar sequence of events can follow any anesthetic, such as the combination of thiopental and a muscle relaxant, which also may cause respiratory depression and respiratory acidosis.

Other changes follow completion of general anesthesia. When a closed system method of administration has been used, its removal is accompanied by reduction in respiratory resistance, reduction in the temperature and humidity of the inspired air, and reduction in the oxygen concentration. Any or all of these could be implicated in the development of hypotension.

Unrecognized or Inadequately Treated Blood Loss

Failure to measure and replace obvious blood loss during extensive operations may contribute to immediate postoperative hypotension.

Blood loss may be hidden temporarily after closure of the thoracic or abdominal cavity, even though drains are in place. Obstruction to outflow through these drains can occur and bleeding is not apparent.

There are certain clinical situations in which prolonged and profound oozing of blood may occur. The lungs, pancreas, prostate and uterus appear to liberate substances which interfere with coagulation. Operations on these organs may therefore be accompanied by generalized oozing from the operative site. Transfusions of large volumes of banked whole blood may also be followed by oozing which is difficult to control. The mechanism of this is still uncertain, but may include the effects of excess citrate, reduced accelerator globulin, reduced platelets, excess heparin-like substances, reduced fibrinogen and fibrinolysis. When massive transfusion is required, the anesthetist must be alert to the possibility of continued, generalized oozing. Use of fresh frozen plasma or fresh (1 to 3 hours) whole blood has proved helpful in improving clot formation, as has the slow intravenous injection of 10 per cent calcium gluconate (0.5 to 1.0 gm).

Embolus

Hypotension following embolization has been discussed (Chapter 20). Although uncommon, pulmonary embolism may be the cause of sudden hypotension at the end of operation. We have been bewildered by several such cases until postmortem findings revealed the cause. Cerebral emboli following mitral commissurotomy commonly cause intractable arterial hypotension. Other emboli may consist of fat or air.

RESPIRATORY SEQUELAE

Hypoxia is a constant threat in the immediate postoperative period. Studies by Comroe and Botelho have indicated the difficulties in the clinical recognition of cyanosis. One must guard against the possibility of unrecognized hypoxia since the signs and symptoms may be elusive. A patient should not be moved from the operating room until the anesthetist is certain about the adequacy of pulmonary ventilation and oxygenation.

Soft Tissue Respiratory Obstruction

Partial or complete respiratory obstruction is a common phenomenon in the immediate postoperative period. In addition to observance of thoracic movement, the palm of the anesthetist's hand should be placed over the patient's mouth and nose to appreciate the exhalation of air with each breath. The adage that noisy breathing is obstructed breath-

ing is valid, and stertor calls attention to the abnormality. With complete obstruction, however, one must rely on the detection of respiratory muscle activity and the absence of movement of air against the sensing hand. Patients must be transported in a manner designed to forestall airway obstruction. The lateral position is safer than the supine in this regard. We have observed death from undetected respiratory obstruction during transportation of anesthetized patients.

Vomiting

Vomiting at the conclusion of operation while the patient is still on the operating table is welcomed by many anesthetists since it may eliminate material which if retained might cause continued nausea and retching. So long as gastric contents are not aspirated into the respiratory tract no harm is done. A high index of suspicion by the anesthetist affords the greatest protection for the patient. Anyone to be anesthetized may vomit. Although swallowing often precedes vomiting, occasionally fluid may well up into the pharynx without prior warning. All of these factors must be remembered as the operation comes to an end.

The 10 to 15 degree head-down tilt recommended by many has little value in the opinion of the authors, for if the mouth or pharynx contains vomitus at the time of an inspiration, inhalation of the material is likely. Nor is excessive stimulation of the pharynx with a suction catheter recommended. This can often be the cause of further gagging and vomiting. A properly functioning suction apparatus must be at hand, but it should be used judiciously. Similarly, an oral airway should not be permitted to remain in place too long, for its presence can stimulate vomiting as anesthesia lightens. When vomiting is anticipated the head should be turned to the side.

If gastric contents enter the respiratory tract we believe that repeated irrigation with 10 to 20 ml. sterile saline introduced through an endotracheal tube is of value. This dilutes irritant acids or alkali. The lungs can quickly absorb large quantities of saline. Unless particulate matter is aspirated, we do not believe that bronchosopic aspiration is superior to the technique just described.

Removal of an Endotracheal Tube

If an endotracheal tube is withdrawn during a light plane of anesthesia, this stimulus may result in closure of the glottis. Laryngospasm can be marked and troublesome, particularly in infants and children. With their high metabolic rate and small residual volume of oxygen,

hypoxia can develop quickly. This complication can be minimized by gentleness during extubation, extubation during deeper planes of anesthesia, or extubation after the patient awakens. Once laryngospasm has developed, frantic efforts to inflate the lungs with oxygen may cause, instead, inflation of the stomach, elevation of the diaphragm, and the likelihood of vomiting. Steady but not excessive pressure to a breathing bag containing oxygen is preferable and is usually sufficient. The intravenous injection of 20 to 40 mg succinylcholine to relax the striated muscle of the larynx followed by manual pulmonary ventilation may be needed. Should neither method be available, insertion of a large bore needle (14 gauge) into the trachea below the cricoid cartilage may be life-saving (Fig. 39). Through this needle one can inject air with a large syringe, or give oxygen via a rubber tube.

Removal of an endotracheal tube may also reveal for the first time that vocal cord paralysis has occurred during operation. Following thyroid surgery signs of respiratory difficulty may indicate such damage. Inspection of the vocal cords will reveal absence of movement with respiration. The surgical team should be alerted to the possible need for tracheostomy.

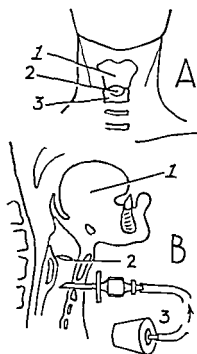


Fig. 39 Needle through cricothyroid membrane for tracheal resuscitation. A. Front view 1 Thyroid cartilage 2 Crico-thyroid membrane 3 Cricoid cartilage B Lateral view 1 Tongue 2 Vocal cords 3 Rubber stopper and tubing for oral insufflation

Diffusion Anoxia

At the end of nitrous oxide anesthesia the pulmonary alveoli and blood contain nitrous oxide and relatively small amounts of nitrogen. With the first inhalation of room air, nitrogen begins to diffuse into the blood and nitrous oxide into the alveoli. Because of its greater solubility in blood, the volume of nitrous oxide available for diffusion outward into the lung is considerably greater than the volume of nitrogen that replaces it in blood. Hence there is a net gain in the gas volume of the lung. With the trachea patent, the lung does not expand, but merely expels the mixture of gases and with it some oxygen. The residual oxygen is diluted and the partial pressure decreased by the outward diffusion of nitrous oxide. Even a few breaths of room air during inhalation of 80 per cent nitrous oxide, as for replacement of a mask or aspiration of the endotracheal tube, can result in a decreased arterial blood oxygen saturation. Prevention of this complication consists in provision of a high oxygen concentration as nitrous oxide is being eliminated. Conceivably this phenomenon might occur at the conclusion of an open ether anesthesia if additional oxygen had not been provided.

Pneumothorax

We have observed pneumothorax following brachial plexus or intercostal nerve block, after rupture of an emphysematous bleb, in association with mediastinal emphysema, during dissections in the neck, when air had not been adequately withdrawn after thoracotomy, or during operations beneath the diaphragm. Examination of the chest must be carried out to identify this abnormality, for should a tension pneumothorax be present, prompt aspiration of the air is essential.

Pulmonary Atelectasis

Collapse of parts of the lungs behind bronchial obstruction must also be considered. This can occur during general anesthesia and remain undiagnosed because the inhalation of a high concentration of oxygen prevents the recognition of hypoxia. When room air is breathed at the conclusion of the operation the effects of atelectasis are unmasked. Again physical diagnosis looms large in the recognition of this entity.

Respiratory Depression from Muscle Relaxants

At the completion of anesthesia which involves use of one of the muscle relaxants respiratory inadequacy may exist. These drugs reduce respiratory minute volume as a result of the neuromuscular blocking action upon intercostal muscles and diaphragm. Effects upon

central regulatory respiratory mechanisms may also prevail. If these pharmacologic actions have not disappeared completely by the end of operation, pulmonary ventilation must be assisted lest hypoxia supervene. This undesirable sequela has occurred with all of the muscle relaxants, even the short-acting members of the group.

Restrictive or Obstructive Dressings

Insufficient alveolar ventilation may be caused by the application of excessively tight surgical dressings after such operations as radical mastectomy. Respiratory obstruction may also be caused by dressings or plaster casts applied about the head and neck. Surgeons must be warned of this possibility as the wound is being covered.

EMERGENCE EXCITEMENT

Restlessness, occasionally progressing to wild delirium, is sometimes seen in the immediate postoperative period. We have noted restlessness in 7 per cent of patients admitted to a recovery room following general anesthesia. The excitement has varied from mild restlessness requiring restraint to delirium of such extent that litters have been overturned. For a discussion of the cause and treatment of this complication, see Chapter 25.

PAIN

Some patients have little postoperative discomfort, in others pain may be intolerable. Pain may contribute to emergence excitement. Pain also sometimes appears to cause hypotension, at least pain relief in some cases has been associated with a rise in blood pressure. Pain, therefore, can be regarded as undesirable, but efforts toward its relief by such drugs as morphine and meperidine must be undertaken cautiously lest excessive depression of respiration and blood pressure and diminution of consciousness follow. A more detailed discussion of this is offered in Chapter 25.

REFERENCES

- Comroe J. H., Jr. and Botelho S. The unreliability of cyanosis in the recognition of arterial anoxemia. *Am J M Sc* 214 1 1947.
Dripps R. D. Abnormal respiratory responses to various curare drugs during surgical anesthesia. Incidence etiology and treatment. *Ann Surg*, 137 145, 1953.

Diffusion Anoxia

At the end of nitrous oxide anesthesia the pulmonary alveoli and blood contain nitrous oxide and relatively small amounts of nitrogen. With the first inhalation of room air, nitrogen begins to diffuse into the blood and nitrous oxide into the alveoli. Because of its greater solubility in blood, the volume of nitrous oxide available for diffusion outward into the lung is considerably greater than the volume of nitrogen that replaces it in blood. Hence there is a net gain in the gas volume of the lung. With the trachea patent, the lung does not expand, but merely expels the mixture of gases and with it some oxygen. The residual oxygen is diluted and the partial pressure decreased by the outward diffusion of nitrous oxide. Even a few breaths of room air during inhalation of 80 per cent nitrous oxide, as for replacement of a mask or aspiration of the endotracheal tube, can result in a decreased arterial blood oxygen saturation. Prevention of this complication consists in provision of a high oxygen concentration as nitrous oxide is being eliminated. Conceivably this phenomenon might occur at the conclusion of an open ether anesthesia if additional oxygen had not been provided.

Pneumothorax

We have observed pneumothorax following brachial plexus or intercostal nerve block, after rupture of an emphysematous bleb, in association with mediastinal emphysema, during dissections in the neck, when air had not been adequately withdrawn after thoracotomy, or during operations beneath the diaphragm. Examination of the chest must be carried out to identify this abnormality, for should a tension pneumothorax be present, prompt aspiration of the air is essential.

Pulmonary Atelectasis

Collapse of parts of the lungs behind bronchial obstruction must also be considered. This can occur during general anesthesia and remain undiagnosed because the inhalation of a high concentration of oxygen prevents the recognition of hypoxia. When room air is breathed at the conclusion of the operation the effects of atelectasis are unmasked. Again physical diagnosis looms large in the recognition of this entity.

Respiratory Depression from Muscle Relaxants

At the completion of anesthesia which involves use of one of the muscle relaxants, respiratory inadequacy may exist. These drugs reduce respiratory minute volume as a result of the neuromuscular blocking action upon intercostal muscles and diaphragm. Effects upon

central regulatory respiratory mechanisms may also prevail. If these pharmacologic actions have not disappeared completely by the end of operation, pulmonary ventilation must be assisted lest hypoxia supervene. This undesirable sequela has occurred with all of the muscle relaxants, even the short acting members of the group.

Restrictive or Obstructive Dressings

Insufficient alveolar ventilation may be caused by the application of excessively tight surgical dressings after such operations as radical mastectomy. Respiratory obstruction may also be caused by dressings or plaster casts applied about the head and neck. Surgeons must be warned of this possibility as the wound is being covered.

EMERGENCE EXCITEMENT

Restlessness, occasionally progressing to wild delirium, is sometimes seen in the immediate postoperative period. We have noted restlessness in 7 per cent of patients admitted to a recovery room following general anesthesia. The excitement has varied from mild restlessness requiring restraint to delirium of such extent that litter has been overturned. For a discussion of the cause and treatment of this complication, see Chapter 25.

PAIN

Some patients have little postoperative discomfort, in others pain may be intolerable. Pain may contribute to emergence excitement. Pain also sometimes appears to cause hypotension, at least pain relief in some cases has been associated with a rise in blood pressure. Pain, therefore, can be regarded as undesirable, but efforts toward its relief by such drugs as morphine and meperidine must be undertaken cautiously lest excessive depression of respiration and blood pressure and diminution of consciousness follow. A more detailed discussion of this is offered in Chapter 25.

REFERENCES

- Comroe J. H., Jr. and Botelho S. The unreliability of cyanosis in the recognition of arterial anoxemia. *Am J M Sc* 214 1 1947
Dripps R. D. Abnormal respiratory responses to various curare drugs during surgical anesthesia. Incidence etiology and treatment. *Ann Surg* 137 145 1953

The Recovery Room

A CRITICAL period in the surgical patient's course is the first few hours after operation. During this time the unconscious or semiconscious patient is completely dependent upon others for his welfare. Even the individual who has received regional anesthesia and is in control of his faculties may require prompt attention, as for hypotension.

A specially staffed and specially equipped room has therefore been established in many hospitals for the care of patients during this period. The benefits resulting from such a room include

- 1 Maximal safety for patients
- 2 Removal of the burden of immediate postanesthetic care from the general nursing staff and economy in distribution of nurses
- 3 Concentration of equipment needed for resuscitation
- 4 Opportunity for the study of problems associated with the immediate postanesthetic period

PERSONNEL

The successful operation of a recovery room depends in great measure upon the skill and devotion of the nursing staff. These individuals must be dedicated to their work, for in few other branches of nursing is attention to detail so vital and the need for immediate, intelligent action so necessary. At any moment a patient's condition may worsen. Instant recognition of this change, proper appraisal of its significance, and prompt application of corrective measures are demanded. The luxury of being able to assign a physician to full-time recovery room duty is denied most hospitals, but anesthetists must be immediately available. In some hospitals student nurses obtain their only experience in the care of unconscious patients while on assignment.

to the recovery room Under close supervision they learn the problems and management of respiratory obstruction, hypotension, emergence excitement and pain

DESIGN AND FACILITIES

There are certain desirable features in the design of a recovery room Lighting should be uniform and of sufficient brightness to permit appraisal of the changes in color of a patient's skin and mucous membranes The nurses' station should command a view of all patients, for constant surveillance by all personnel attached to a recovery room is essential During the immediate postanesthetic period separation of the sexes is not necessary Nor do we favor cubicles or routine use of curtains unless an attendant is assigned to each patient At least two telephones are required so that in emergencies an open line is more apt to be available A call system permitting the nurses to summon additional help is useful A special area for assembling, cleaning or sterilizing equipment, and for storing apparatus infrequently used, should adjoin the patient area rather than be a part of it

Resuscitation requires that oxygen and suction outlets be provided for each patient Masks should be on hand to provide inspired concentrations of oxygen greater than the 40 per cent afforded by a properly placed nasopharyngeal catheter Mechanical ventilators providing intermittent positive pressure as well as those offering a positive-negative phase should be available All types and sizes of pharyngeal airways, endotracheal tubes, laryngoscopes, a bronchoscope, and tracheostomy sets are essential Few drugs are useful as respiratory stimulants, but bronchodilating agents should be at hand for patients with asthma or with bronchoconstriction from other causes Isoproterenol (Isuprel) and aminophylline can be used for these conditions

For support of the circulation normal saline solution, glucose in water and one of the plasma expanders must be provided Several units of type O, Rh negative and Rh positive whole blood should be stored in the recovery room refrigerator for emergency transfusions These are for use until properly cross-matched blood is made available In addition to the usual intravenous sets, provision must be made for introducing blood under pressure Venous cut-down trays, cannulas and plastic tubing of various sizes are also part of resuscitative equipment To support the blood pressure methoxamine (Vasoxyl), phenylephrine (Neo-synephrine), ephedrine and norepinephrine (Levophed) are most often used Hydrocortisone for intravenous use is essential

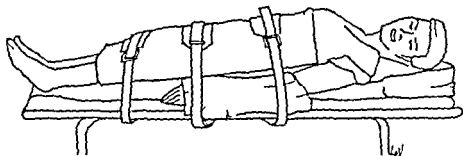


Fig 40 Broad restraining strap for emergence restlessness and excitement.

should adrenal cortical deficiency exist. An electrical defibrillating apparatus is indicated—and an external artificial pacemaker. A cardiac kit with knife and thoracic retractors should also be readily available.

Heart failure may occur in the recovery room. Venous tourniquets should be available, as well as facilities for the rapid removal of blood from a vein. Digitoxin and more rapidly acting drugs of this type and anti-arrhythmic substances such as quinidine, procaine amide and molar sodium lactate must be stored. An electrocardiograph should be at hand.

Facilities for the management of all types of drainage tubes are needed, for example, gastrointestinal suction apparatus, bladder irrigating sets, under-water chest drainage units and sump pumps.

Broad restraining straps (Fig 40), wristlets, arm boards and side boards for beds may be needed for the management of severe restlessness. Narcotics, barbiturates, chlorpromazine and apomorphine must be available. For overdosage of the opiates, nalorphine or levallorphan is often necessary. A locked cabinet for narcotics is needed.

Standard equipment such as blood pressure cuffs and manometers, stethoscopes, syringes and needles, thermometers, urinary bladder catheters, emesis basins, urinals and bedpans must be at hand. A supply of linen and blankets should be kept in stock.

RECORDS

An accurate record of the patient's course in the recovery room must be kept. The vital signs (blood pressure, pulse, respiratory rate and body temperature) should be recorded as they are taken. All therapeutic measures should be listed as given. Pertinent observations of the patient's appearance and reactions should be noted. This record becomes a part of the permanent hospital record and is of value to personnel who assume subsequent responsibility for the patient, providing as it does a

summary of the early postoperative period. A copy of this record should also be attached to the anesthetist's copy of the anesthesia sheet.

Sequelae which must be anticipated in the immediate postoperative period have been listed in an earlier chapter. In addition to these, one may watch for hyperpyrexia, shaking chills or a lowering of body temperature. A marked and prolonged elevation of blood pressure may follow the use of pressor drugs, or the absorption of bladder irrigating fluid during transurethral operations. Gastric dilatation may occur. Embolic phenomena in the lungs or brain may develop. Disturbing cardiac rhythms may be noted. Cardiac arrest may occur. These and other challenging situations require the best in diagnostic acumen.

RELEASE OF PATIENTS

The most reliable criterion for discharging a patient from the recovery room is the good judgment of the anesthetist and the nursing staff. The following appraisals are valuable:

1. Stability of vital signs
2. Lack of the danger of aspiration of vomitus
3. Orientation of the patient as to time and place
4. Ability to maintain a safe position in bed, e.g., optimal position for drainage or adequate respiration

Discharge of some patients will depend upon the primary reason for their admission, that is, outpatients must be kept until they can walk without dizziness or nausea. Patients who have received spinal anesthesia should be detained until the level of anesthesia begins to recede and arterial blood pressure is stable.

INTENSIVE THERAPY UNIT

There is no agreement as to the ideal length of stay in a specialized area such as we have described. The facilities of certain recovery rooms have been expanded to include an intensive therapy unit for several days of postoperative care. Desperately ill medical as well as surgical patients can be admitted to this area. Beds rather than litters are more suitable for this purpose. Kitchen facilities are needed and a considerably more complex unit must be envisaged. There is no doubt that the critically ill individual will receive the best care, day and night, in such a highly organized section of the hospital. In some institutions the recovery room and the recovery ward are separate but coordinated units, the former being responsible only for patients during the early postoperative hours.

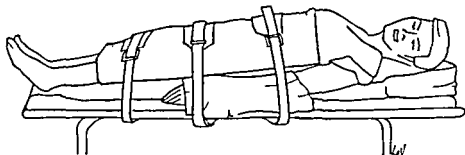


Fig 40 Broad restraining strap for emergence restlessness and excitement.

should adrenal cortical deficiency exist. An electrical defibrillating apparatus is indicated—and an external artificial pacemaker. A cardiac kit with knife and thoracic retractors should also be readily available.

Heart failure may occur in the recovery room. Venous tourniquets should be available, as well as facilities for the rapid removal of blood from a vein. *Digitoxin* and more rapidly acting drugs of this type and anti-arrhythmic substances such as quinidine, procaine amide and molar sodium lactate must be stored. An electrocardiograph should be at hand.

Facilities for the management of all types of drainage tubes are needed, for example, gastrointestinal suction apparatus, bladder irrigating sets, under-water chest drainage units and sump pumps.

Broad restraining straps (Fig 40), wristlets, arm boards and side boards for beds may be needed for the management of severe restlessness. Narcotics, barbiturates, chlorpromazine and apomorphine must be available. For overdosage of the opiates, nalorphine or levallorphan is often necessary. A locked cabinet for narcotics is needed.

Standard equipment such as blood pressure cuffs and manometers, stethoscopes, syringes and needles, thermometers, urinary bladder catheters, emesis basins, urinals and bedpans must be at hand. A supply of linen and blankets should be kept in stock.

RECORDS

An accurate record of the patient's course in the recovery room must be kept. The vital signs (blood pressure, pulse, respiratory rate and body temperature) should be recorded as they are taken. All therapeutic measures should be listed as given. Pertinent observations of the patient's appearance and reactions should be noted. This record becomes a part of the permanent hospital record and is of value to personnel who assume subsequent responsibility for the patient, providing as it does a

Treatment of Immediate Postoperative Pain and Excitement

PAIN is perceived when patients who have been operated upon recover consciousness following general anesthesia, or regain normal sensation after local or spinal anesthesia. Those who have recovered completely from general anesthesia, and those who have received local anesthesia, can be questioned about the magnitude and location of pain, and an accurate appraisal made. Those who awaken more slowly and who have not regained control of their actions may indicate pain only by restlessness or excitement. There are in addition other causes of excitement in the immediate postoperative period. Both pain and excitement pose hazards to the patient. Likewise, the treatment of these conditions is not without danger. An approach to this dilemma will be offered in this chapter.

IMMEDIATE POSTOPERATIVE PAIN

Nearly all patients who have had an operation experience pain as they recover from anesthesia. The early manifestation of pain is dependent upon many factors. Narcotics used for preanesthetic medication, or those given intermittently as supplements to general anesthetics, or by single injection prior to the completion of operation, diminish postoperative pain. When narcotics are given for preanesthetic medication, little analgesia remains after long operations. Operations involving extensive dissection and those with incisions affected by

SUMMARY

A recovery room has become an essential part of an anesthesia and surgical service. Centralization of personnel and equipment has been associated with a reduction of postoperative mortality and morbidity. Only in such an area can the patient be brought through the early postoperative period with maximum safety. Deserving emphasis also is the teaching value of this type of unit. Student nurses, medical students, interns and residents gain much from the opportunities to observe the reactions and participate in the treatment of patients in the immediate postoperative period.

RESTLESSNESS AND EXCITEMENT

Restlessness or excitement may arise from a combination of several factors which include antecedent psychomotor disturbances, pain, the prior administration of drugs, hypoxia, and other ill defined factors. In order to treat effectively a patient exhibiting these symptoms, the underlying cause must be recognized. The apparent causes will be discussed first.

Patients who suffer from psychomotor disturbances, as for example mania, agitation or chronic alcoholism, are likely to show postoperative excitement. The anesthetist should recognize this possibility during the preanesthetic visit. Patients who have been or who are under the care of a psychiatrist should be suspect. Those who are unduly concerned, those fearful of the findings at operation, and those who tell the anesthetist that they are cowards and cannot stand pain are more likely to be excited postoperatively.

Pain may be the cause of excitement. If the patient is sufficiently awake to complain of pain, the cause of the excitement is obvious. If he is still partly anesthetized, excitement may be a manifestation of pain uncontrolled by normal cerebral inhibition. Pain may be of the incisional type, or it may arise from distention of a hollow viscus, as for example the urinary bladder.

Certain drugs increase the likelihood of development of postoperative excitement. Scopolamine, given to normal individuals, often produces hallucinations, flight of ideas and agitation. We have noted an increased incidence of emergence excitement in patients to whom scopolamine alone, or in combination with a barbiturate, has been given for pre-anesthetic medication. It does not appear as commonly if a narcotic has been used with scopolamine. The incidence of excitement is less if atropine is substituted for the scopolamine. Phenothiazine derivatives, particularly promethazine (Phenergan) alone or in combination with barbiturates, may cause restlessness before as well as after anesthesia. When these drugs are combined with a narcotic, restlessness is less likely to be observed. Certain individuals, particularly women, may also respond to narcotics with excitement.

Mild degrees of oxygen lack lead to increased motor activity and restlessness. Hypoxia mimics the second stage of anesthesia. Excitement due to hypoxia should be suspected especially in patients who have had thoracic operations, or head and neck or upper abdominal procedures. It may also appear in the presence of pneumothorax, tracheal collapse and vocal cord paralysis, or with undue respiratory

respiratory movements, as in thoracic and upper abdominal procedures, are apt to be followed by severe pain. Severe pain is a common feature after perineal procedures which are accompanied by rectal sphincter spasm, such as hemorrhoidectomy. Complaints are more likely in those with preoperative psychogenic disorders or those with emotional instability, or when the operation has caused unusually great concern. Reactivity to pain differs greatly among individuals. The very young and the elderly usually show less reaction. Emotionally stable and philosophical individuals probably experience equally severe pain but are less demonstrative.

The reactivity of patients to pain appears to be related to the degree of anesthetic-induced cerebral cortical depression. In the presence of such depression, normal inhibitions are removed and the individual has less self-control than when consciousness is present. In this condition, restlessness may be an outstanding response to pain. Objectively, pain often causes a rise in blood pressure as well as a rise in heart and respiratory rate. However, pain may lead occasionally to a reduction of blood pressure. In the latter instance, pain relief is followed by a rise in blood pressure.

The treatment of immediate postoperative pain in patients who have had general anesthesia must be carefully gauged. An overdose of narcotic can lead to reanesthetization and circulatory or respiratory depression. The first postoperative dose of narcotic should be a small one. A good rule to follow is that the initial dose should be half that anticipated for the patient on the first postoperative day. For example, if 10 mg of morphine were ordered, the first dose should be 5 mg.

In 1876 Claude Bernard noted that a small dose of morphine would return patients to the anesthetized state after they had nearly recovered from chloroform anesthesia. Small intramuscular doses of narcotics given to the patient still partially under the influence of general anesthetics will often provide pain relief and a quieting effect. We have found 5 mg of morphine or 25 mg of meperidine given intramuscularly satisfactory for this purpose in about 50 per cent of a series of postoperative patients. Hypotension and respiratory depression are not common with these doses but become disturbing with larger amounts. It is safer to give a second small dose of narcotic than to administer a large one initially. If a narcotic analgesic has been administered following anesthesia, patients should be carefully observed. Occasionally the combined effects of the anesthetic and the analgesic produce further circulatory and respiratory depression.

2.5 mg to 5.0 mg doses. Meperidine may be administered in similar fashion in 12.5 mg to 25 mg doses. The intravenous dose of chlorpromazine should not exceed 2.5 mg but it may be repeated at intervals as indicated. Apomorphine may be injected in subemetic doses (1.0 to 1.5 mg diluted in 5 to 10 ml of saline) and injected slowly intravenously.

A final method of controlling postoperative excitement is the administration of cerebral stimulants, such as caffeine and sodium benzoate or nikethamide given intravenously. The rationale is to hasten awakening and return of cerebral cortical control of emotions by increasing cerebral blood flow and counteracting the effect of anesthetics on the brain. These medications may cause hypotension, retching and vomiting. Nikethamide may occasionally cause tonic or clonic muscle spasms. Intravenous injections should be made slowly and with careful observation. We have seen prolonged hypotension and cerebral vascular damage after the intravenous injection of 10 mg of chlorpromazine given to treat emergence delirium.

Fortunately, as the anesthetics are eliminated and self-control is re-established, agitation lessens and the patient becomes quiet. Time, therefore, is on one's side. The experience, however, may be a traumatic one, not only for the patient but for his attendants as well.

depression following general anesthesia and the use of relaxant drugs. Finally hypotension may contribute to excitation through cerebral hypoxia.

There is a group of patients in whom the cause of excitement is not clear. Such patients have most commonly had general anesthesia. Excitation has been noted after all general anesthetics, including the intravenous barbiturates, but seems to be most common after cyclopropane alone or when given in combination with diethyl ether. It often develops in the muscular person, in those accustomed to an excessive alcoholic intake, and more often in the male. The background in all these cases may be concealed anxiety, with the added effects of pre-anesthetic medication and anesthesia.

Various reasons have been propounded for this phenomenon. These include carbon dioxide retention, an emergence excitement stage comparable to that of induction, an action on the brain of the catecholamines, epinephrine and norepinephrine or their metabolic products liberated during anesthesia, and prolonged maintenance of one body position. The latter factor is important in some patients. We have seen patients in whom excitement or restlessness has promptly disappeared when the patient was turned to another position.

The prophylaxis of postoperative excitement should be apparent from some of the aforementioned etiologic factors. Those patients who are emotionally disturbed or who have psychomotor disturbances should receive extra preanesthetic sedatives. A small dose of narcotic administered prior to the conclusion of anesthesia may assist in diminishing emergence excitement. We have found it wise not to use scopolamine alone or in combination with a barbiturate in patients in whom this complication is likely to occur. We do not advocate the use of promethazine, because of the higher incidence of agitation before and after anesthesia. If a patient gives a history of excitement following a particular narcotic, we omit all narcotics or substitute one of a different chemical configuration. Measures designed to treat hypoxia or hypotension will prevent excitement based on these factors.

Should excitement appear, the following treatment can be applied. If the patient has been in one position upon the operating table for a long period of time, it is well to change this position in the bed or on the litter. If pain is a factor, a small dose of narcotic is usually effective. If hypoxia appears to be the underlying cause, the inhalation of a high concentration of oxygen will usually quiet the patient. In other situations, morphine or meperidine, chlorpromazine or apomorphine is usually useful. Morphine may be injected slowly intravenously in

actions in the patient. Mineral and vegetable oils, ethers, esters, oxidizing acids and hydrocarbon solvents cause swelling, tackiness and more or less rapid destruction of the rubber. Copper and manganese lead to catalytic decomposition. Phenols and cresols are not only destructive but may subsequently lead to cutaneous burns if impregnated in the rubber. Water, alkalis and salts of mercury do little harm. Plastics are particularly susceptible to destruction by heat and strong chemicals.

CLEANSING BEFORE STERILIZATION

The contaminated parts of anesthetic apparatus include face masks, airways, laryngoscope blades, suction catheters, delivery tubes, reservoir bags, and, in the anesthesia machine, directional valves, carbon dioxide absorbers and certain types of vaporization bottles. Before sterilization, apparatus should be cleaned of gross dirt. Although soap and hot water are used commonly for this purpose, ordinary soap is not bacteriostatic. The process of scrubbing equipment may be a hazard to personnel if pathogenic bacteria are present. For this reason it has been suggested that disinfection of all apparatus be carried out initially in solutions of Wescodyne, a detergent-iodine complex (100 parts per million), which is tuberculocidal and viricidal. Scrubbing can be performed with this solution without personal hazard. The lumina of endotracheal and pharyngeal airways can be cleansed with tight fitting, stiff bristled malleable brushes. Pipe cleaners can be used for finer apertures. Other equipment should be scrubbed with brushes, paying particular attention to crevices and angles that might accumulate dirt and secretions. This is probably the single most important aspect of care of equipment. The brushes themselves should be periodically sterilized. Suction catheters and metal suction tips should be rinsed with water under pressure. Adhesive tape and oily lubricants should be removed with waste ether. At the termination of mechanical cleansing the parts should be thoroughly rinsed in tap water. Certain parts of the apparatus such as reservoir bags, delivery tubes and face masks, if not used in patients with obvious transmissible disease, may then be hung up to drain and dry, and can be used again.

CHEMICAL STERILIZATION

Ordinary soaps are not bacteriostatic but soaps containing detergents and bacteriostatics are available. The best known commercially are Dial and Gamophen, each containing hexachlorophene. These are not

Cleaning and Sterilization of Equipment Used for Inhalation Anesthesia

ANESTHETIC equipment used for inhalation anesthesia should be so cared for that the possibility of transmission of infectious disease from one patient to another is minimal. The esthetic aspect of using clean equipment need not be discussed, and deterioration of equipment that might result from proper cleansing must be only a secondary consideration. The literature concerning transmission of disease through anesthetic apparatus is confusing at best. A careful epidemiologic study has never been carried out. It is important to note that pathogenic bacteria, including the tubercle bacillus, have been cultured from anesthetic equipment at the termination of anesthesia in infected persons. For this reason we have adopted the practice of treating anesthetic equipment as if it were going to be used on ourselves. There should be sufficient equipment in an anesthetic department so that parts can be properly cleaned while others are in use.

DETERIORATION OF EQUIPMENT

Heat and chemical treatment are the two means of sterilizing apparatus. Both techniques if conscientiously employed lead inevitably to deterioration, particularly of rubber parts. Since equipment is expensive, the least harmful method should be adopted. Sanders has pointed out that the constituents of natural rubber, the porosity, and the chemicals used in manufacture render rubber susceptible to deterioration. Some of these constituents may be a source of allergic re-

is impermeable to bacteria and the transparency allows for easy identification of the contents

SPECIAL PRECAUTIONS IN TUBERCULOSIS

In this disease or other conditions with virulent pathogens everything used during the anesthetic must be sterilized. If closed system anesthesia is selected, the to-and-fro technique should be used, since a circle filter is much more difficult to sterilize. We have adopted the practice of setting aside equipment for use only in patients with pulmonary tuberculosis and not for any others. If the patient has a positive sputum he should wear a mask before induction of anesthesia and the mask should be reapplied afterward. He should be segregated from other patients. The anesthetist should wear a gown and some may prefer to wear gloves. At the completion of the procedure the anesthetist's gown is placed in a "dirty linen" bag to be autoclaved along with other material from the operating room. Gloves and soda lime are decontaminated in Lysol solution, and the soda lime then discarded. All metal parts of the anesthetic equipment are autoclaved. Rubber parts are boiled in water for 15 minutes. The anesthesia machine should be scrubbed with a tuberculocidal chemical. The following disinfectants have been shown to be effective against the tubercle bacillus: Ethyl alcohol, 70 per cent, isopropyl alcohol, 70 per cent, cresol solution saponated U S P XIII, orthophenyl phenol, p tert amylphenol 2 per cent (O-Syl, Lehn and Fink).

REFERENCES

- Gross G L. Decontamination of anesthesia apparatus. *Anesthesiology* 16 903 1955
Joseph J M. Disease transmission by inefficiently sanitized anesthetizing apparatus. *J A M A* 149 1196, 1952
Livingstone H et al. Cross infection from anesthetic face masks. *Surgery* 9 433, 1941
McDonald W L, Welch H J and Keet, J E. Antisepsis of endotracheal tubes and face masks. *Anesthesiology* 16 206, 1955
Sanders R D, and Aikins, J P. Notes on the Characteristics, Composition and Care of Rubber Materials Used in Medical Goods. Wilmington, Delaware

as effective as a liquid preparation, pHisoHex, which contains pHiso-derm, a detergent and 3 per cent hexachlorophene pHisoHex, however, may leave a film of lanolin on rubber parts McDonald has shown that a one-minute vigorous scrub with pHisoHex employing 40 to 100 strokes will sterilize face masks, airways and laryngoscope blades against ordinary pathogens Although hexachlorophene is highly bacteriostatic for gram-positive organisms, it is less so for the tubercle bacillus and gram-negative bacteria pHisoHex is hypoallergenic and non-irritating in contrast to most other chemicals

Gross has described a simple, rapid and non-injurious technique of sterilization After gross cleansing in soap and water, a five minute period of soaking in 1 1000 aqueous Zephiran (benzalkonium) eliminated practically all pathogenic bacteria Cultures for the tubercle bacillus were not made Soaking in 1 1000 aqueous Zephiran for 12 hours produced sterility for all common pathogens This technique can be employed routinely for all apparatus, rubber and metal, that comes in intimate contact with the patient's respiratory tract, being careful to change the Zephiran solution daily Zephiran is inexpensive, rapid in action, non-irritating and hypoallergenic as far as we know

HEAT STERILIZATION

Heat sterilization causes more deterioration of rubber and plastics than the chemicals already mentioned but eliminates the possibility of allergic reactions and irritation from the latter It is probably the most reliable method of sterilization when applicable Rubber and metal parts may be boiled in water for 15 minutes for practical purposes Mask cushions should be deflated and endotracheal cuffs first removed Both rubber, with a greater chance of destruction and metal parts can be autoclaved, the usual time being 15 minutes at 250° C and 15 lbs pressure When a circle filter has been contaminated, it should be disassembled and all parts autoclaved

STORAGE OF CLEAN EQUIPMENT

When apparatus has been cleaned or sterilized it should not be re-contaminated by careless storage or repeated handling before use In this connection it is worth noting that the tops of anesthetic machines and work tables must not be wantonly soiled with dirty apparatus, as clean equipment will become contaminated Clean equipment can be kept in sterile towels or sterile containers The circular type of container is good to maintain a proper shape for endotracheal tubes Transparent plastic tubing has been used for storing catheters and tubes The plastic

V

Special Topics

Inadequacy of Coronary Arterial Blood Flow There may be a decreased supply of blood to the myocardium or an increased demand for blood which the coronary arteries cannot meet. In the former instance, one can implicate hypotension from hemorrhage, spinal, epidural or local anesthesia, overdosage of preanesthetic medication or general anesthetics, traction reflexes, the onset of Stokes-Adams attacks, or raised airway pressure. So far as increased demand is concerned, the injection of cardiac stimulants, for example epinephrine, may cause such an increase in cardiac work and need for increased blood flow that the supply falls behind the demand and myocardial ischemia results.

The heart itself can be depressed to the point of cessation of activity, either through the effects of *reflexes* or by the action of *drugs*. Reflexes arising from the carotid sinus, from pulmonary vagal endings of the von Bezold type, or from manipulations at the hilum of the lung are examples of the first mechanism. Digitalis, quinidine, procaine amide, narcotics and general anesthetic drugs can be included in the second category.

Oxygen lack, whatever the cause, is a potent factor in the development of cardiac arrest. Respiratory obstruction or depression is a common precipitating event.

Miscellaneous causes include electrocution, hypothermia, pulmonary or cerebral embolus, or manipulation of the heart during operation. The list is not complete but is illustrative.

Diagnosis

The following signs are suggestive of cardiac arrest

- 1 Absence of radial and carotid pulses
- 2 Inaudible heart sounds
- 3 Sudden pallor or cyanosis
- 4 Sudden pupillary dilatation
- 5 Respiratory standstill or apneustic gasps
- 6 Absence of bleeding and dark blood in the operative field
- 7 Electrocardiographic evidence of asystole

Most of these signs are unreliable. They may be present because of severe hypotension as well as of cardiac arrest. We therefore recommend the following course of action if one is uncertain of the diagnosis. Unless one can see the heart, or can palpate the abdominal aorta (through an incision already made), the only positive way of diagnosing cardiac arrest is by opening the left chest. In the rare instance in which a thoracotomy is performed and the heart is found to be beating feebly,

Cardiac Resuscitation

CESSATION of cardiac activity or fibrillation of the ventricles occurring in the operating room can usually be successfully treated without subsequent brain damage if a correct diagnosis is made and if therapy is instituted at once. Success is less likely if such a catastrophe occurs outside of the operating room, but an increasing number of dramatic restorations are being reported under these conditions. It would seem worth while making the effort anywhere if the diagnosis is made promptly. So-called cardiac resuscitation, however, should not be attempted when success cannot be hoped for, i.e., in terminal conditions, massive hemorrhage, and the like.

The anesthetist may witness cardiac arrest during induction of anesthesia, during operation, or in the immediate postoperative period. It is more likely to occur in elderly patients, in those with previous paroxysmal cardiac arrhythmias, in heart block, in digitalis overdosage, following massive hemorrhage, and during operations on the heart. A high index of suspicion, for whatever reason, makes it mandatory that the anesthetist start out with an electrocardiograph, an artificial pacemaker, large gauge intravenous needles and a full complement of drugs to which we shall presently refer. It cannot be stressed too often that successful resuscitation demands the instant and simultaneous application of a number of appropriate measures. To hesitate in applying these is to court disaster. A plan of approach to the problem must be made beforehand and implemented immediately when the occasion arises.

Factors in Cardiac Arrest

There are several common factors which may precipitate cardiac arrest

heart may produce myocardial irritability sufficient to cause ventricular fibrillation. The primary value of drugs lies in increasing the force and/or rate of cardiac contraction, once the heart has begun to beat spontaneously. To increase force, epinephrine, in doses of 1 or 2 ml of 1:10,000 solution, is most potent, but may be followed by ventricular fibrillation. A more controllable stimulant is isoproterenol (Isuprel) in doses of 0.1 to 0.2 mg. A third drug is calcium chloride, 5 to 10 ml of a 10 per cent solution. These compounds are injected into the left ventricle and moved through the coronary arterial system via manual compression of the heart.

If the cardiac rate is slow after spontaneous activity has resumed, atropine sulfate in doses of 0.4 mg intravenously may release vagal tone and cause a more satisfactory rate of contraction.

While manual systole is being carried on, an electrocardiogram should be taken since the electrical complexes of the heart can be a useful guide to drug therapy. If the electrocardiogram is that of the so-called "dying heart," i.e., showing a "disorganized" appearance with widened and slurred QRS complexes, molar sodium lactate solution administered intravenously may prove helpful. One hundred ml of this solution may be required, the risk of providing an excessive sodium ion level seems acceptable in light of our present knowledge.

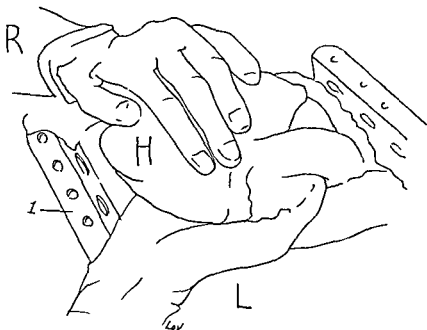


Fig 41 Method of performing manual systole (after M. Codding). R, Right hand; L, Left hand; H, Heart; I, Rib spreader.

little harm is done providing oxygenation of the lungs can be carried out. This has happened to us on two occasions, in each the decision to open the chest was based upon the absence of detectable blood pressure and peripheral pulses. Although the heart was beating weakly at first sight, in both instances it stopped within a few moments of exposure.

TREATMENT

Cardiac Arrest

The instant cardiac arrest is suspected and as preparations are made to open the chest, one can pound on the precordium with one's fist or needle the heart through the chest wall. Both of these maneuvers can be done quickly and are suggested because of the presence of a greater degree of cardiac irritability in the early phases of arrest. While these are attempted, artificial respiration with oxygen must be begun via a face mask—intubation of the trachea can wait.

The incision should be made in the fourth or fifth left intercostal space. It should not extend to the sternum lest the internal mammary artery be cut, nor, in one's haste, should one incise the lung. The pericardium need not be opened at the start. The heart is cradled in the operator's hand, and the ventricles compressed between thumb and fingers of one hand or fingers of two hands (Fig. 41). Manual compression of the heart (manual systole) can serve as an effective means of moving blood to the cerebral and coronary arteries. In response to each compression a pulse should become palpable in the carotid artery. If this is not felt, compression is inadequate. Pupillary constriction replacing dilatation is a good sign of cerebral circulation. Cardiac compression should be applied at the rate of 40 or 50 per minute in order to ensure sufficient movement of blood. This is about as fast as one can go without cramping of the hand.

There must be sufficient fluid available for cardiac output during intermittent manual systole. Blood may be pooled in the periphery when the heart stops. It is necessary therefore to provide fluids intravenously or intra-arterially. The rate of injection must be rapid if the cardiac chambers are to fill between compressions. This may involve cutting down upon a vein and inserting a large-bore needle or cannula. Any type of parenteral fluid can be used in this emergency—volume is the essential item. Once the heart begins to fill satisfactorily, beware of overloading the circulation.

Before drugs are used, the cardiac muscle should be made as pink as possible through manual systole and pulmonary ventilation with oxygen. This is vital, for the injection of chemical agents into a hypoxic

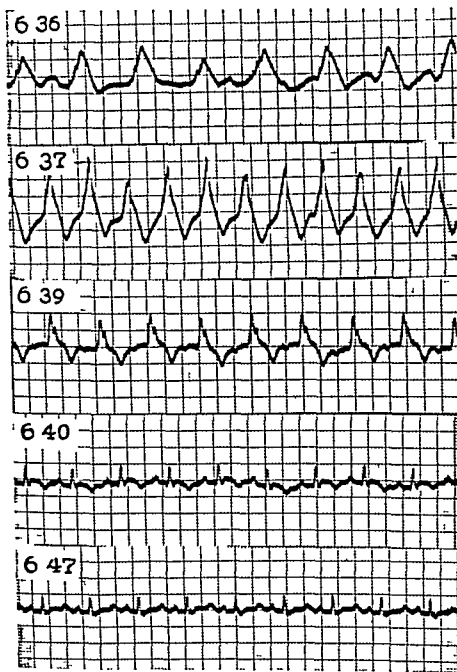


Fig 42. The effect of molar sodium lactate intravenously upon the electrocardiogram of a patient with ventricular fibrillation.

6 36—Wide and slurred ventricular responses one minute after start of molar sodium lactate together with manual systole

6 37—Spontaneous cardiac action.

6 39—Norepinephrine added intravenously

6 40 and 6 47—Continued improvement of ventricular complexes

The Anesthetist's Role The anesthetist is usually the first to suspect cardiac arrest. If convinced of the diagnosis, he should insist that the chest be opened. He should oxygenate the blood during the crisis and counteract the pneumothorax. Immediate intubation of the trachea is unnecessary and may cause delay in oxygenation if there are difficulties in introducing a tube. The anesthetist should palpate a peripheral artery, usually the carotid, or ask someone else to do it, to test adequacy of manual compression. He directs the rate of infusion of fluids to ensure adequate filling of the heart. Finally, the anesthetist makes careful notes, if he can, as to the time and course of events. Another person can be designated as timekeeper if one is available. If the patient's surgeon has had no experience with cardiac resuscitation or is unwilling to make the move, the anesthetist should make it without hesitation!

Ventricular Fibrillation

If the ventricles are fibrillating the first measure to take is to oxygenate the myocardium via manual systole. With oxygen supplied to the lungs and circulation through the coronary arterial system established through compression of the heart, the myocardium should lose its dusky color and become pink. Electrical defibrillation should then be attempted. Shocks of 160 to 180 volts and 0.1 to 0.2 second's duration are applied through disc electrodes. The larger the mass of the heart, the greater the voltage required. Defibrillation of a badly diseased heart is difficult to accomplish. Fortunately mere passage of an electrical current through the patient's body will not explode a potentially explosive mixture of anesthetic gases. To avoid electrocution, surgeon and anesthetist must avoid contact with the patient as the shocks are applied. If an electrical defibrillator is not available, asystole can be produced by injection of 6 to 8 mEq potassium chloride into the root of the aorta. The aorta should be briefly compressed distally and the drug carried through the coronary arterial system by manual systole.

Defibrillation is usually followed by asystole, the treatment for which has just been outlined. If the heart resumes beating with sufficient force to provide an adequate blood pressure, and if the electrical complex is reasonably normal, no further steps need be taken. However, successful defibrillation may be followed by a spontaneous reversion to ventricular fibrillation. If the heart rate is very slow, e.g., 20 per minute or less, and the ventricular electrical complexes are wide and slurred, molar sodium lactate solution given intravenously may be of value (Fig. 42).

Principles of Pediatric Anesthesia

PREPARATION FOR OPERATION

Psychologic Aspects

Infants and children require special attention prior to operation. They may be unable to understand the reason for being in the hospital and why the separation from family has taken place. Inadequate preparation for anesthesia, or inept handling, can produce psychic trauma which may last for years. The highest incidence of this complication appears in the one to two year age group, then declines gradually and levels off at seven to eight years. These sequelae can be minimized by the anesthetist's visiting the child when a parent is present. If the child understands that the anesthetist is his parent's friend and that he will see him again that afternoon or the following morning, he will be reassured. Psychic trauma can also be minimized by a display of confidence and lack of apprehension on the part of the parents. The importance of the preceding statement can and should be imparted to the father and mother by the referring physician.

Peanesthetic Medication

Medication administered at the correct interval before the child leaves the ward will alleviate apprehension. Drugs should be given according to the suggestions in Table 11, one hour prior to the time of induction of anesthesia. Weight in the average child is a better guide to dosage than age. Negro children should be given the next higher dose of belladonna drug because of a tendency to form excessive secretions in the respiratory tract. Apprehensive children should receive the next higher dose of narcotic or barbiturate. In warm or humid weather the

PROGNOSIS

Damage to the central nervous system from ischemia is the most important determining factor in the prognosis when restoration of cardiac action has been accomplished. If periods of anoxia have preceded the complete cessation of cerebral blood flow associated with cardiac arrest, the brain's ability to survive the latter insult may be reduced below the four minute figure generally accepted. There are few measures known that can prolong this survival time, that is, the four minutes of total ischemia. Hypothermia can be protective. Perhaps general anesthesia, by reducing cellular metabolism, may afford slight protection.

Signs which presage recovery include prompt reversal of pupillary dilatation and reaction to light, return of spontaneous respiration, cessation of decerebrate movements, and rapid return of consciousness and orientation. Under these circumstances complete recovery can be practically guaranteed. With severe central nervous system damage there is evidence of irritability and cell destruction, such as convulsions, fever and persistent coma. Further anoxia from repeated convulsions should be prevented with barbiturates and Dilantin. The prognosis here must be guarded. The longer these conditions persist the more hopeless the outlook. Serial electroencephalograms can provide a quantitative means of the degree of damage and improvement.

APPRAISAL

Cardiac resuscitation has saved and will save many lives. It is a dramatic type of therapy which should be thoroughly understood *before* one is called upon to apply it. Careful planning is essential to its success. If possible, practice in manual systole and defibrillation should be sought in the animal laboratory to provide a bit of self-confidence. Conviction of the necessity for prompt diagnosis and immediate effective action is the key to success.

Table 12 Approximate Respiratory and Circulatory Data in Children*

WEIGHT (lbs)	AVERAGE TIDAL VOLUME (ml)	AVERAGE MINUTE VOLUME (ml)	SIZE OF ENDO TRACHEAL TUBE O D (mm)	LENGTH OF ENDO TRACHEAL TUBE (cm)	RESPIRA TORY RATE IN SLEEP	BLOOD VOLUME (ml)	AVERAGE HEMO GLOBIN (gm)	BLOOD PRESSURE	AVERAGE PULSE RATE (per minute) Asleep Anesthetized
8	16-21	750-1175	4 6	10	20-80	450-500	15-21	78/50	130
10	23	800-1270	4 6	10	20-80	550	11-12	90/60	130
12	41	1200-1650	5 3	10	40	600	10-11	95/60	130
15	51	1750-1950	6 0	11	38	750	11-12	100/70	120
21	78	2650-3000	6 3	12 5	38	1000	12	100/70	112
								(4 5 cm cuff)	
31	138	4410	7 6	14 0	32	1500	12	100/70	95
36	140	4480	8 0	15 5	32	1800	13	110/70	90
45	215	5375	8 6	17 0	25	2250	13		85
60	231	5540	9 6	18 5	24	3000	14		85
77	355	7810	10 0	20 0	22	3500	14		80
85	395	7900	10 6	22 0	20	4250	14	115/75	80
								(9 cm cuff)	
									80-150
									80-150
									75-140
									75-120
									70-110
									70-110

O D = Outside Diameter

* Data of Margery VanN Demings, M D

Table 11 Preadnesthetic Medication for Children

AGE	WEIGHT IN POUNDS	MORPHINE	SECobarbital OR Pentobarbital	Scopolamine
		Subcutaneously or Intramuscularly	Orally Rectally or Intramuscularly	Subcutaneously or Intramuscularly
Up to 2 mos	7-10 lbs	—	—	gr 1/800
2-3 months	10-12 lbs	—	—	gr 1/600
3-4 months	12-14 lbs	—	—	gr 1/600
4-7 months	14-16 lbs	—	—	gr 1/600
7-11 months	16-19 lbs	—	—	gr 1/600
11-18 months	19-24 lbs	gr 1/96	gr 1/4	gr 1/600
18-24 months	24-27 lbs	gr 1/72	gr 1/4	gr 1/400
2-3 years	27-30 lbs	gr 1/64	gr 1/4	gr 1/400
3-5 years	30-40 lbs	gr 1/48	gr 1/2	gr 1/400
5-8 years	40-55 lbs	gr 1/32	gr 1/2	gr 1/300
8-10 years	55-65 lbs	gr 1/24	gr 1	gr 1/200
10-12 years	65-80 lbs	gr 1/16	gr 1	gr 1/200
12-14 years	80-90 lbs	gr 1/8	gr 1-1/2	gr 1/150

To make up	Morphine 1/96	Dissolve gr 1/8 in 12 ml	SDW*	Give 10 ml
	1/72	gr 1/8 in 9 ml		10 ml
	1/64	gr 1/8 in 8 ml		10 ml
	1/48	gr 1/8 in 6 ml		10 ml

* SDW = Sterile Distilled Water

The apothecary system has been used in this table because tablets of morphine and scopolamine are generally available only in that system

dose of belladonna drug should be reduced since interference with sweating minimizes loss of heat by evaporation. Scopolamine is preferred to atropine since it is a better drying agent and offers additional central sedation. Barbiturates, as well as narcotics, may be given with the belladonna drug as a single intramuscular injection. Barbiturates taken by mouth can be dissolved in a spoonful of syrup and given prior to the hypodermic injection. It is unnecessary and, we believe, hazardous to use narcotics in children who are to be anesthetized by the 'open drop' method (see reasons on p 61).

ANESTHETIC MANAGEMENT

Those who anesthetize infants and children should familiarize themselves with the unique characteristics of respiration and circulation in the child, listed in Table 12. There should be an awareness of the special

Measurement of Blood Pressure) Many apply a wide cuff and obtain readings which are falsely low. Similarly, cuffs of narrow width may produce abnormally high readings. Several sizes of blood pressure cuff should therefore be available. The proper size is that which will fit the upper arm without encroaching on the axilla or antecubital space (Fig. 43). Unless the cuff covers most of the arm length it is too small. There must be available several sizes of stethoscope bell, the smallest, for infants, being about 1 cm. in diameter.

Induction

Anesthesia should be induced in a manner to avoid further apprehension, crying or struggling. If the child is asleep from the preanesthetic medication, it may be possible to induce anesthesia by allowing a mixture of nitrous oxide and cyclopropane to flow over the face by gravity. The mask is gradually lowered and when sufficient anesthesia has been established, the mask is approximated to the face.

When the child is awake and cooperative there are several methods of inducing anesthesia subtly, such as "blowing up the balloon," or "counting out loud." Distraction is the essence here. With a little ingenuity anesthesia can be induced satisfactorily in the majority of children. Any anesthetic agent and most techniques applicable to the adult can be used for the child.

Induction of anesthesia in children can be accomplished on the ward by the rectal instillation of a basal dose of thiopental or Avertin. This should be considered only when personnel are available for constant observation of the patient while he is unconscious. An enema should be given the evening before operation or at least four hours preceding the instillation of the anesthetic agent. Avertin fluid ordinarily should not be employed in doses of over 30 to 40 mg. of tribromethanol per pound of body weight (60 to 90 mg. per kilogram). Thiopental in a 10 per cent solution can be injected through a rectal catheter on the following dosage scale where thiopental is to be the sole anesthetic agent, 20 mg. per pound, preanesthetically for basal narcosis, 10 mg. per pound, for the treatment of recurrent convulsions, 5 mg. per pound. The means for resuscitation should always be at hand when rectal anesthetics are administered.

Maintenance

Pulmonary Ventilation and Temperature Regulation Steps must be taken to assure adequate pulmonary ventilation, to prevent heat retention, and to satisfy fluid requirements. Techniques of anesthesia

Table 13 Daily Fluid Requirements

WEIGHT (pounds)	H ₂ O ml /lb
5-25	80-50
25-90	50-20
90-135	25-20

problems of fluid balance (Table 13) Greater detail concerning these matters can be ascertained from any of several textbooks dealing with pediatric anesthesia Vital functions of the child should be observed, as in adults Blood pressure, pulse and respiratory rates should be recorded at regular intervals A careful accounting of blood lost and fluids administered should be made In long operations body temperature should be measured at least at 30 minute intervals to detect fever A reasonable reduction in body temperature is less to be feared and is often desirable

Estimations of blood pressure in infants and small children are accurate only if the proper size of cuff is used (see chapter on the

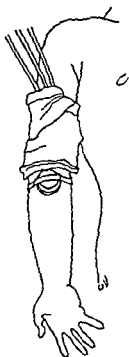


Fig 43 Proper placement of blood pressure cuff in the child Stethoscope in antecubital space

should be performed and a cannula or plastic tubing inserted into the vein. A transfusion set incorporating stopcocks and a syringe to inject blood rapidly should be available (Fig. 44).

Endotracheal Anesthesia

Endotracheal anesthesia is not always essential in infants or children but the technique is used more frequently than in adults to minimize respiratory dead space and to improve pulmonary ventilation. When this method is elected, great care should be exercised to prevent trauma to soft tissue and to prevent laryngeal edema. Slight degrees of edema may lead to respiratory obstruction, stridor and consequent hypoxia. The principles involved in endotracheal anesthesia are those of surgically clean equipment, the avoidance of oversized endotracheal tubes, and gentleness in manipulation.

There are certain anatomic differences between the infant and adult larynx. These differences have practical implications.

- 1 The vocal cords in the infant are at the level of cervical vertebral bodies 2 to 4 as compared with cervical bodies 5 to 6 in the adult. This means that in the infant the angle formed by laryngoscope and trachea may be greater than in the adult, leading to difficulty in intubation.

- 2 The angle formed by the epiglottis and vocal cords is more acute in the infant. Exposure of the infant larynx is thus more of a problem.

- 3 The epiglottis of the infant is short, and therefore more difficult to elevate with the laryngoscope blade.

- 4 The epiglottis is "U" shaped in the infant and flat in the adult.

- 5 In the infant the anterior attachment of the vocal cords is more inferiorly placed than the posterior attachment. The opposite is true in the adult. A curved endotracheal tube is more apt to 'hang up' on the anterior commissure in the infant for this reason.

- 6 The larynx is funnel-shaped in the infant and tends to be cylindrical in the adult. The funnel configuration is formed by the narrowing of the larynx at the cricoid cartilage. The diameter of the airway at this point may be less than at the vocal cords. Hence, an endotracheal tube that may be accommodated by the glottic opening may not pass into the trachea through the cricoid ring. Forcing a tube under these circumstances will lead to laryngeal irritation and edema of the laryngeal mucous membrane.

Prior to extubation of the trachea, a catheter should be passed into the stomach to reduce the risk of postoperative vomiting and aspiration. Gentle suction should be applied to remove fluid and to relieve gastric

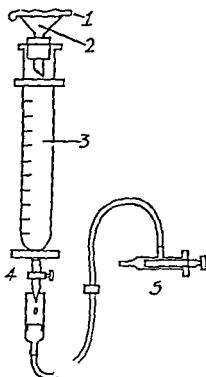


Fig. 44 Transfusion set for infants (Courtesy Robert Smith) 1 Gauze covering. 2 Funnel 3 Burette 4 Stopcock. 5 Injection syringe

that predispose to heat retention should be avoided. Non-rebreathing, open or semi-closed systems are preferable. Anesthesia with the open technique should be accompanied by insufflation of oxygen at a rate of 0.5 to 1.0 liter per minute beneath the mask to ensure adequate oxygen in the inspired air and to facilitate the elimination of carbon dioxide. If the body temperature begins to rise during the course of a long procedure, this should be countered by covered ice bags placed in the axillae and between the thighs. A minimum of drapes should cover the patient.

Blood Transfusion Blood loss must be carefully observed and measured, and the need for replacement anticipated. During major operations, blood should be replaced as the loss occurs. Seemingly negligible blood losses in an infant may be of major importance in relation to the blood volume. Thus, a loss of 10 ml. in a ten pound infant may equal a 200 ml. loss in an adult. Blood may be infused at the rate of 10 ml. per pound of body weight per hour plus the estimated blood loss during the first hour of operation. Thereafter the measured loss may be replaced volume for volume. In major operations on small children reliance should not be placed on venipuncture alone, but a phlebotomy

Anesthesia for Ambulatory Patients

General Preparations

Instructions to outpatients should be precise and preferably in written form. Explanation should be made for each directive. Adult outpatients should be accompanied by a relative or friend to escort them home, and patients should be cautioned not to drive a car. These people must be forewarned that it may be necessary to remain in the hospital for several hours postoperatively. Physicians referring children for outpatient operations should give the parents a list of instructions similar to that reproduced here.

INSTRUCTIONS TO PARENTS

Your child has been scheduled for an operation and will be put to sleep for this procedure. The safety of the anesthesia will depend in part upon your following these instructions. In the hospital the doctors and nurses take care of these details, but because your child will not come into the hospital until immediately before the operation, you must assume this responsibility.

The following instructions have been prepared for children of different ages. They are given to be sure that your child has an empty stomach at the time of operation. If a child has food in its stomach and vomits during anesthesia, some of the food may become lodged in the windpipe and be quite dangerous.

Infants If your child has been receiving a bottle as part of his usual meal, the last feeding should be at least three hours, but not longer than four hours, before the operation. Only milk, formula, or strained fruit juice should be given—no baby food or solid food.

Children Eating Regular Meals If the operation is in the morning, give only one of the following at least three hours before the scheduled operation: a glass of strained fruit juice, or tea with milk and sugar, or skimmed milk for breakfast. AVOID ALL SOLID FOODS, CEREAL, AND WHOLE MILK.

If the operation is in the afternoon, your child may have breakfast no later than 9:00 A.M. This should consist of strained fruit juice and a cooked cereal such as

distention. Caution must be exercised in performing tracheobronchial toilet at the conclusion of the anesthetic. Strong or prolonged suction must be avoided and the endotracheal tube should not be withdrawn with the suctioning catheter protruding, because of the possibility of producing laryngospasm. This complication can produce profound anoxia.

CONVULSIONS

Children who have fever, acidosis and dehydration are prone to convulse when anesthetized with general anesthetics. Prior to operation, these patients should be given 5 per cent glucose in water intravenously and the infusion should be continued during the anesthetic. When possible, some form of local or regional anesthesia or a combination of barbiturate, nitrous oxide and muscle relaxant should be used. In any case, the retention of carbon dioxide and of heat should be prevented since these predispose further to convulsions. When premonitory twitching or frank convulsions occur, small amounts (25 to 50 mg.) of thiopental must be injected intravenously and the lungs inflated with oxygen.

REFERENCES

- Eckenhoff, J. E. Preanesthetic sedation of children. *A.M.A. Arch. Otolaryngol.* 57:411 1953.
Eckenhoff, J. E. Some anatomic considerations of the infant larynx influencing endotracheal anesthesia. *Anesthesiology* 12:401 1951.
Lough, M. D. and Bolton, M. K. *Pediatric Anesthesia*. The Macmillan Co. New York, 1948.
Smith, R. M. Blood replacement in thoracic surgery for children. *J.A.M.A.* 167:1124 1956.
Stephen, C. R. *Elements of Pediatric Anesthesia*. Charles C. Thomas, Springfield, Illinois 1954.

Anesthesia for Ambulatory Patients

General Preparations

Instructions to outpatients should be precise and preferably in written form. Explanation should be made for each directive. Adult outpatients should be accompanied by a relative or friend to escort them home, and patients should be cautioned not to drive a car. These people must be forewarned that it may be necessary to remain in the hospital for several hours postoperatively. Physicians referring children for outpatient operations should give the parents a list of instructions similar to that reproduced here.

INSTRUCTIONS TO PARENTS

Your child has been scheduled for an operation and will be put to sleep for this procedure. The safety of the anesthesia will depend, in part, upon your following these instructions. In the hospital the doctors and nurses take care of these details, but because your child will not come into the hospital until immediately before the operation, you must assume this responsibility.

The following instructions have been prepared for children of different ages. They are given to be sure that your child has an empty stomach at the time of operation. If a child has food in its stomach and vomits during anesthesia, some of the food may become lodged in the windpipe and be quite dangerous.

Infants. If your child has been receiving a bottle as part of his usual meal, the last feeding should be at least three hours, but not longer than four hours, before the operation. Only milk, formula, or strained fruit juice should be given—no baby food or solid food.

Children Eating Regular Meals. If the operation is in the morning, give only one of the following at least three hours before the scheduled operation: a glass of strained fruit juice, or tea with milk and sugar, or skimmed milk for breakfast. AVOID ALL SOLID FOODS, CEREAL, AND WHOLE MILK.

If the operation is in the afternoon, your child may have breakfast no later than 9:00 A.M. This should consist of strained fruit juice and a cooked cereal such as

Fanna oatmeal or Cream of Wheat, with skimmed milk. Do not give solid foods such as eggs, corn flakes or other types of cold cereal. Give nothing by mouth (this includes both liquids and solids) after breakfast.

Children can face unpleasantness if told the truth. Do not make up false stories about what will happen at the hospital. The simple truth told by an understanding parent is much better than fairy tales.

Bring your child to the hospital not later than one hour prior to the time of operation so that he can be examined and given an injection to make him sleepy. The wait before and after operation is difficult for parents, but necessary for the safe conduct of recovery from the anesthetic. A waiting room is provided for the parents' convenience.

Outpatients should have had a history taken and a physical examination performed. A urinalysis and hemoglobin determination are mandatory. These data should be available to the anesthesiologist. An anesthetic and operative permit should have been signed.

Adult ambulatory patients should omit the meal prior to the time of scheduled operation even if local anesthesia is to be used. Nothing should be ingested during the four to six hours prior to operation. The nausea and retching that may occur during or following local or general anesthesia is less troublesome if the stomach is empty. Narcotics should be omitted because of the long duration of action and the tendency to produce hypotension, vertigo, fainting, nausea and vomiting when the patient sits up or walks. Atropine should be given intramuscularly 45 minutes before operation in those patients scheduled for general anesthesia. Barbiturates may be indicated for nervous or excitable patients. The shorter acting drugs, secobarbital or pentobarbital, taken by mouth are preferable under these circumstances. Their administration may make for a smoother anesthetic course, although recovery from anesthesia can be delayed.

Outpatients scheduled for general anesthesia should appear at the hospital one hour prior to the scheduled time of operation. They will then be given preanesthetic medication and asked to rest quietly until the anesthesiologist and surgeon are ready. Clothing should be removed and hospital attire worn to avoid soiling. The patient should be asked to void and also to remove dentures prior to anesthetization.

Choice of Agents and Technique

The ideal choice for ambulatory patients is local anesthesia, utilizing infiltration, field block or regional block techniques. Short-acting local anesthetic agents and minimal concentrations are preferred. If epinephrine is to be used with local anesthetics, the optimum concentration (1:200,000) must be employed since excessive quantities produce many

of the so-called "reactions" to local anesthetics (nervousness, palpitation, tremor and vertigo)

The general anesthetic agents of choice are those that are rapidly taken up and eliminated. Nitrous oxide, ethylene, cyclopropane, trichlorethylene, divinyl ether and ethyl chloride satisfy these requirements. Because of lack of preanesthetic medication, nitrous oxide and ethylene are often inadequate in providing a sufficient depth of anesthesia. These drugs, however, can be more satisfactorily used in combination with trichlorethylene or divinyl ether. For operative procedures requiring only analgesia, trichlorethylene can be administered in combination with air by an inhaler. Divinyl ether or ethyl chloride can be used with the "open drop" method, but the latter drug should be used with care to avoid sudden elevations in blood concentration and circulatory depression. Its use is best confined to short operations, such as myringotomy or reduction of a dislocation. In our hands, cyclopropane has proved to be an effective way of anesthetizing outpatients when a more potent agent was required. Thiopental is inadvisable because postoperative recovery may be delayed.

Proper restraints, suction and resuscitative equipment should be at hand during use of these anesthetics and patients should be placed in the recovery room immediately postoperatively. The period of observation should be sufficiently long to rule out the possibility of delayed complications. These include such sequelae as tension pneumothorax after brachial or intercostal block, difficulty in vision due to atropine, or hemorrhage from the operative site. The patient should not be allowed to leave until discharged by the anesthetist and then only after he has demonstrated his ability to walk about without difficulty.

Obstetric Anesthesia and Infant Resuscitation

OBSTETRIC patients are usually normal healthy individuals. Because of this, and the fact that the majority of women are delivered of infants without incident, obstetric anesthesia often fails to challenge the anesthetist. Anesthesia for obstetrics has therefore not infrequently been relegated to the less experienced and is often poorly done.

Three aspects of obstetrics deserve the anesthetist's particular consideration. These are the dependence of the infant's life upon the events that occur during parturition, the unpredictable time of delivery, and the fact that certain complications of pregnancy appear suddenly and without warning.

Dependence of the Infant's Survival upon Events Occurring During Labor

Prenatal or neonatal death may result from (1) Oversedation of the mother in an attempt to relieve the pain of labor. The excessive use of narcotics during labor depresses the vital processes of infant as well as mother. (2) Maternal hypotension which is not uncommon following the use of continuous caudal, lumbar epidural or spinal anesthesia for labor and delivery, may lead to suboxygenation of the fetus. (3) Maternal asphyxia or hypoxia. The infant normally has a low arterial oxygen saturation. Windle has shown that the respiratory center of the animal fetus is more likely to be depressed by oxygen lack than is that of the mature animal. It has also been shown that hypercapnia produces hyperventilation in the mother and little or no change in the fetus. The effects of suboxygenation and hypercapnia may therefore be less

well tolerated by the infant than the mother (4) Mechanical obstetrical factors (such as nuchal cord, premature placental separation and prolonged labor) may interfere with infant circulation and oxygenation

The Unpredictable Time of Delivery

This involves the following (1) Administration of anesthesia to a patient who may have recently eaten Aspiration of vomitus during general anesthesia is a major cause of maternal death (2) The need for rapid administration of anesthetics to relieve the pain of the final stages of labor To quiet the patient, high concentrations of anesthetic may have to be given hurriedly with resultant respiratory and circulatory depression (3) Unavailability of attendants This may result in the administration of anesthetics by unskilled individuals as well as the attendance upon the mother of inexperienced obstetrical personnel

The Complications of Pregnancy

(1) Hemorrhage This is a common complication due to placenta previa, placenta abruptio, uterine atony or cervical tears Great skill and speed of action on the part of anesthetists and obstetricians alike may be required to save the lives of mother and infant in these emergencies (2) Abnormal positions of the infant These may require profound anesthetization of the mother to produce uterine relaxation for podalic version and breech extraction

ANALGESIA AND AMNESIA DURING EARLY PHASES OF LABOR

A complete discussion of this aspect of obstetrics is beyond the scope of this volume A few points of interest will, however, be considered Opiates, chiefly meperidine (Demerol), and short-acting barbiturates such as secobarbital, are the drugs commonly used for analgesia and amnesia during labor Narcotics depress the respiration of both infant and mother Such depression is potentially more dangerous to the infant than that from the inhalation anesthetic agents, since the latter can be recovered from the infant by pulmonary ventilation after delivery Even low concentrations of inhalation anesthetics may potentiate the depressant effect of narcotic analgesics Narcotics should be given to the mother sparingly and should be avoided in the 60 to 90 minutes prior to delivery Barbiturates and scopolamine are less depressant to mother and infant However, agitation commonly results and occasionally one encounters a patient who responds to these two drugs with unusually deep sedation

The possibility of obtaining analgesia during the first stage of labor with inhalant agents should not be overlooked. Trichlorethylene can be self-administered by the cooperative and intelligent patient. Nitrous oxide in concentrations of 50 to 75 per cent with oxygen can be administered by an anesthetist. To be effective, this must be administered as a uterine contraction begins. The anesthetist should therefore palpate the mother's abdominal wall to detect the onset of contraction or ask the patient to warn him when she feels a contraction first begin. Neither of these methods is apt to be effective if the mother has been given large doses of narcotics and sedatives, since she may have lost the ability to cooperate. Narcotics can be omitted or the dose reduced if analgesia with trichlorethylene or nitrous oxide is provided.

NORMAL VAGINAL DELIVERY

General Anesthesia

Inhalation anesthesia is the most controllable method of obstetrical anesthesia. It is usually available and can be given to the patient who is crowning, moving or uncooperative. It is the most reliable from the patient's standpoint. While any of the inhalation agents may be chosen, we prefer nitrous oxide and oxygen with or without small amounts of ether. Deep anesthesia is not required for normal vaginal delivery. Four liters of nitrous oxide with one and a half liters oxygen per minute are often adequate. Such a mixture is administered preferably in a non-rebreathing system to aid in elimination of nitrogen. This facilitates the uptake of nitrous oxide and hastens induction. Since the majority of patients will have had narcotics for pain relief, analgesia may be reached within a few minutes. The anesthetist can judge the adequacy of analgesia or anesthesia by observing the patient's response to the pain of uterine contraction or episiotomy. When the patient lies quietly during either of these, conditions are suitable. Both obstetrician and anesthetist should remember that several minutes are required for the development of maximal analgesia. Both must also realize that during light anesthesia it may be difficult to avoid movement of the patient in response to pain. When nitrous oxide has been used in the manner described, the patients are usually awake shortly after the termination of the delivery and repair. This minimizes postanesthetic complications.

If suitable conditions cannot be provided with nitrous oxide, ether should be added. Deep ether anesthesia is to be avoided since it causes uterine relaxation and excessive bleeding. Once the infant's head has been delivered, the ether can usually be stopped. Minute to minute observation is necessary, however, and a return to ether may be required for repair of the episiotomy.

We do not advocate the use of intravenous barbiturates to produce general anesthesia for vaginal delivery. While ideal from the point of view of rapidity of induction, they pose a threat of respiratory depression to the infant. Should delivery be delayed beyond a few minutes after drug injection, the infant's circulation may pick up enough barbiturate to prevent the early establishment of respiration following birth.

Whenever general anesthesia is to be given for obstetrics, the usual precautions must be taken. The most common problem during general anesthesia for vaginal delivery is vomiting. Aspiration of vomitus with resultant obstruction of the airway or chemical pneumonitis is common and often the cause of maternal death. Patients who have recently eaten or in whom there is suspicion that the stomach still contains food should not be given general anesthesia. Spinal anesthesia or regional block is preferable. A technique that has proved to have merit in our clinic has been the provision of analgesia with 50 per cent nitrous oxide and oxygen and the infiltration of the line of incision for the episiotomy with 1 per cent procaine. Nitrous oxide can then be discontinued during the repair of the episiotomy.

Spinal Anesthesia

The technique for the administration of spinal anesthesia is described in Chapter 15. Only a block of the sacral nerves need be obtained for the second and third stages. Lumbar puncture may be performed with the patient sitting or in the lateral decubitus position. The drug should not be injected while a uterine contraction is in progress, since injection at this time may result in a high level of anesthesia. Hyperbaric solutions are usually used. If the lateral position is utilized, the table should be in reverse Trendelenburg when the patient is placed supine or several pillows may be placed under the head and shoulders to produce the same effect. This will reduce the likelihood of a high ascent of a hyperbaric solution.

Certain problems may arise during the administration of or following spinal anesthesia for vaginal delivery: (1) The patient is less able to flex the spine for lumbar puncture because of the size of the abdomen. (2) Because of the increased abdominal pressure, and probable reduction in volume of cerebrospinal fluid, hyperbaric solutions often rise rapidly and high levels of anesthesia may result. Therefore, one should keep the amount of drug and total volume injected at a minimum (5 mg tetracaine with 0.5 ml. 10 per cent dextrose). (3) If the level of motor anesthesia rises too high, uterine action may cease and labor will be

retarded (4) Hypotension frequently follows spinal anesthesia under these circumstances, the blood pressure must be carefully observed (5) The incidence of headache following lumbar puncture with ordinary needles for spinal anesthesia may be quite high (over 20 per cent)

Single Injection or Continuous Caudal Block

For the details of the technique of these procedures the student should consult a text on regional anesthesia Continuous caudal anesthesia can be used throughout labor, whereas spinal anesthesia is used only for the terminal stages When continuous caudal anesthesia is employed properly, certain advantages accrue (1) The dose of depressant drugs administered to the mother for pain relief is reduced (2) There is little need to administer general anesthetics during delivery, thereby reducing the possibility of infant depression (3) Maternal convalescence is smoother because of the diminished need for narcotics, barbiturates and anesthetics during labor and delivery (4) There is a psychologic value in having the mother alert and able to appreciate the arrival of her child

The disadvantages of continuous caudal are the following (1) The method is time-consuming in administration and technically more difficult than spinal or general anesthesia Anatomic variations of the sacrum are considerable (2) It is applicable only if sufficient time exists prior to delivery to perform the procedure and obtain analgesia This usually requires a minimum of twenty minutes (3) Occasionally succeeding doses of local anesthetics are less effective than the initial dose (4) Hypotension may result from the block of sympathetic nerves (5) Infection is a possibility

The local anesthetics used for continuous caudal include piperocaine (Metycaine) 1.5 per cent, lidocaine (Xylocaine) 1.0 per cent to 1.5 per cent, or procaine 1.5 per cent to 2 per cent When the patient is ready to deliver, the final dose of local anesthetic is administered, the needle or catheter withdrawn and the patient moved with care onto the delivery table Close observation is mandatory at this time as hypotension can occur with the final injection or with movement of the patient

Pudendal Nerve Block

The technique of this block will not be considered It can be performed either by obstetrician or anesthetist Good perineal anesthesia and relaxation can be obtained but relief from the discomfort of uterine contractions is not provided It is the method of choice for patients who

have recently eaten and those in whom a general or spinal anesthetic is contraindicated. It provides the same advantages of caudal anesthesia save for incompleteness of pain relief. It is technically simpler than a caudal block and is less likely to produce hypotension or result in infection. When combined with nitrous oxide analgesia, it can provide complete comfort for the mother.

ANESTHESIA FOR PODALIC VERSION

Anesthesia for this procedure differs from that already discussed because of the need to obtain a deeper level of anesthesia rapidly to allow for abdominal wall and uterine relaxation, and for intra-uterine manipulation. Once the manipulation is complete, there must be a quick return to a light plane of anesthesia to permit the return of uterine tone and to prevent excessive bleeding. Cyclopropane and oxygen are valuable for this purpose.

ANESTHESIA FOR CESAREAN SECTION

Elective

Mothers scheduled for elective cesarean section usually receive a belladonna drug with or without a barbiturate for preanesthetic medication. We believe spinal anesthesia to be the anesthetic technique of choice because of the absence of infant depression. The original objections voiced by obstetricians were based upon the lack of recognition and inadequate treatment of hypotension. Improved anesthetic care makes these objections untenable. The precautions mentioned in spinal anesthesia for vaginal delivery should be observed, although the patients do not have uterine contractions. A sensory level of anesthesia above T-8 is not needed. This can almost always be obtained with 10 mg. of tetracaine, or less, with equal parts of 10 per cent dextrose. It is common for a high level of anesthesia to result because of the increased intra-abdominal pressure. The blood pressure should be observed frequently to detect early hypotension. In the event of a lowered arterial blood pressure, treatment should be instituted promptly.

Once the infant has been delivered, the mother may be given a narcotic intramuscularly or, if necessary, small doses of an intravenous barbiturate may be used. Immediately following delivery it is customary to give an intravenous or intramuscular injection of ergotrate or Pitocin to promote uterine contraction. The intramuscular route is preferred since hypertension or vomiting may follow the intravenous injection.

When general anesthesia is used, cyclopropane with or without ether

supplementation is preferable. Every effort should be made to minimize the time interval between induction of anesthesia and delivery of the infant to avoid depression of the infant's respiration. Deep anesthesia should be avoided and muscle relaxants are rarely necessary.

Controversy exists as to the use of Pitocin in the presence of cyclopropane anesthesia. Concern is expressed that the combination may result in ventricular fibrillation because of coronary arterial constriction produced by the pituitrin which may be present in Pitocin. Data to support this belief are lacking. While we do not advocate the routine use of this combination, we have no hesitancy in using Pitocin in well oxygenated patients receiving cyclopropane anesthesia, if the obstetrician believes the drug must be used.

Emergency

Cesarean section may be scheduled as an emergency because of fetal distress, uterine inertia, pelvic disproportion or maternal hemorrhage. Under these circumstances an intravenous infusion should be started, with a large bore needle. Blood must be available as well as a means for rapid administration. Oxygen should be administered to the mother until the operating room and surgeons are ready. Anesthesia should be induced preferably with cyclopropane because of the speed with which anesthesia can be obtained. While some favor the nitrous oxide-oxygen-ether sequence, we believe the prolonged induction with this combination to be a disadvantage. Operation should commence as soon as possible, and the infant should be speedily delivered, no longer than three to four minutes should elapse between the start of anesthesia and the incision. Abdominal relaxation is generally not required because of the lack of tone of the abdominal muscles. When possible two anesthesiologists should be present, one to care for the mother, the other for the infant.

The incidence of respiratory difficulty is higher in infants born by cesarean section than in those delivered vaginally. Gastric suction has revealed that the stomachs of the former infants contain more fluid than do those delivered through the pelvis. It has been postulated that cesarean section babies may aspirate amniotic fluid at the time of birth, or swallow this fluid, later regurgitating it and inhaling it into the lungs. For these reasons aspiration of the stomach is recommended for all infants born by cesarean section. This maneuver must not, however, take precedence over the treatment of respiratory depression or obstruction.

ASPHYXIA NEONATORUM

The anesthetist can assist in the early detection of this condition by affixing a stethoscope over the abdomen where the fetal heart sounds are best heard. He can determine the heart rate from minute to minute and can warn the obstetrician of slowing which is a good indication of fetal distress. Should a slow rate be detected, the inspired oxygen given the mother should be increased. If the heart rate does not change after additional oxygen, the infant should be speedily delivered.

The most common causes of asphyxia neonatorum are (1) prematurity, (2) narcotics given to the mother during labor, (3) overdose of anesthetic agents, and (4) trauma of delivery. The anesthetist can do little about the first and last. When an infant is delivered and fails to breathe shortly thereafter, the following plan of therapy should be adopted.

Secretions, amniotic fluid and debris should be aspirated from the oropharynx with a rubber bulb suction device.

Oxygen should be administered with intermittent positive pressure from a bag and mask or with a resuscitator. Care must be taken to avoid overinflation of the lungs or distention of the stomach. Generally 20 to 30 cm. of water is the maximum safe inspiratory pressure. Since the infant thorax is in a position of maximum inspiration, the chest wall will not move much with inflation of the lungs. Excessive movement suggests overinflation.

If it is obvious that the lungs are being ventilated, tracheal intubation is not indicated. If there is doubt, the glottis should be carefully exposed with a laryngoscope, a small suction catheter inserted into the trachea and secretions aspirated. Often this maneuver will initiate a cough or start respiration. If not, one should insert a sterile 14 F endotracheal tube and continue with inflation of the lungs. With this airway overinflation becomes a greater possibility than ever before. Intubation of the newborn infant's trachea may lead to laryngeal edema, it should not be taken lightly. If, however, the indication for intubation is present, one should not hesitate to proceed. Artificial respiration may be continued indefinitely by this means.

Continued failure of spontaneous respiration suggests depression of the infant owing to narcotics given the mother, to birth trauma, or to a congenital anomaly. If there is reason to suspect the former, nalorphine (Nalline) 0.2 to 0.5 mg. or levallorphan (Lorfan) 0.05 to 0.10 mg. may be injected into the umbilical vein. A total of at least 2 ml. of solution should be used to be certain that the drug gets into the infant's circula-

tion If narcotization is the etiologic factor, the child should begin to breathe within several minutes If overdose of anesthetic, birth trauma or congenital anomaly is the cause, the antagonists will be of no avail

If the heart rate becomes slow and feeble, ephedrine 5 mg or caffeine sodium benzoate 50 mg injected intravenously may be helpful

The following procedures are not recommended (1) Slapping, contrast baths, and other means of external stimulation (2) Inhalation of carbon dioxide mixtures—the infant's arterial carbon dioxide tension is already above normal (3) Pentylenetetrazol (Metrazol), nikethamide (Coramine), alpha-lobeline and other analeptics, all these are contraindicated in the presence of hypoxia

Obstetric anesthesia can be interesting if the anesthetist participates in the entire care of the mother and infant rather than confining his efforts to the technical administration of an anesthetic during the rush of delivery The proper management of analgesia with narcotics, hypnotics and inhalational anesthetics is challenging The correct choice and administration of the anesthetic are demanding The successful resuscitation of a cyanotic limp newborn is a satisfying experience

REFERENCES

- Eckenhoff J E Hoffman G L and Funderburg L W N Allyl normorphine An antagonist to neonatal narcosis produced by sedation of the parturient *Am J Obster & Gynecol* 65 1269 1953
- Hershenson B B *Obstetrical Anesthesia Its Principles and Practice* Charles C Thomas Springfield Ill, 1955
- Little, D M Hampton, L J and White M L Asphyxia neonatorum The syndrome its prevention and its treatment *Anesthesiology* 13 518 1952
- Lull C B and Hingson, R A *Control of Pain in Childbirth* J B Lippincott Co, Philadelphia 1945
- Snyder F F *Obstetric Analgesia and Anesthesia* W B Saunders Co Philadelphia 1949
- Wyle, W D *The Practical Management of Pain in Labour* Year Book Publishers Chicago 1953

The Management of Narcotic Poisoning

THE CARE of a patient suffering from an overdose of a narcotic is essentially that of an individual during general anesthesia. An anesthetist, therefore, can contribute much to the management of these patients. Those concerned with the admission of comatose individuals to hospitals should be apprised of the wisdom and urgency of seeking help. Anesthetists should respond to the call with all possible speed and with a rational plan of treatment.

Depressant drugs *per se* seldom cause death unless taken in massive doses. On the other hand, prolonged suboxygenation or arterial hypotension secondary to the action of these substances can lead to irreparable damage or death. Establishment of adequate pulmonary ventilation and support of the circulation therefore take precedence over all else. Once this has been accomplished, efforts at differential diagnosis can be begun, the remainder of the therapeutic program instituted and a guarded prognosis given. The anesthetist called for such an emergency should take with him an emergency or "E" kit kept in readiness at all times, together with a bellows resuscitator for maintenance of ventilation. These items are illustrated in Figures 45 and 46.

URGENT TREATMENT

Establishment of Adequate Pulmonary Ventilation

A patent airway must be maintained through correction of soft tissue obstruction and removal of secretions from the air passages. If

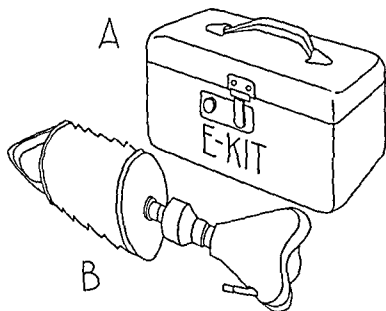


Fig 45 A Emergency kit B Kreiselman resuscitator

tracheal reflexes are present and soft tissue obstruction exists, an oropharyngeal airway will probably suffice. When pharyngeal and laryngeal reflexes are diminished or absent, the trachea should be intubated (35 French uncuffed endotracheal tube in adults, or smaller as indicated). A catheter for aspirating secretions should be passed through this tube every hour and the tube changed every twelve hours. It is removed when pharyngeal and tracheal reflexes return.

Respiratory tidal exchange and minute volume must be adequate. The former can be measured for accurate appraisal. Apnea, shallow respirations, cyanosis, subnormal saturation of hemoglobin or hypercapnia as shown by arterial blood analyses indicates the need for artificial respiration. A mechanical respirator or manual rhythmic compression of an anesthetic bag will suffice. During coma from any cause, oxygen should be administered. If respirations are adequate, 3 to 4 liters per minute of oxygen can be administered through a catheter placed within the endotracheal tube. In the absence of an endotracheal tube, 6 to 8 liters per minute of oxygen can be administered via a nasopharyngeal catheter.

To facilitate drainage of secretions from the respiratory tract, the lateral decubitus position is best, with the head slightly lower than the feet. Lowering of the head of the bed more than 5 degrees may interfere with respiration through elevation of the diaphragm. The patient

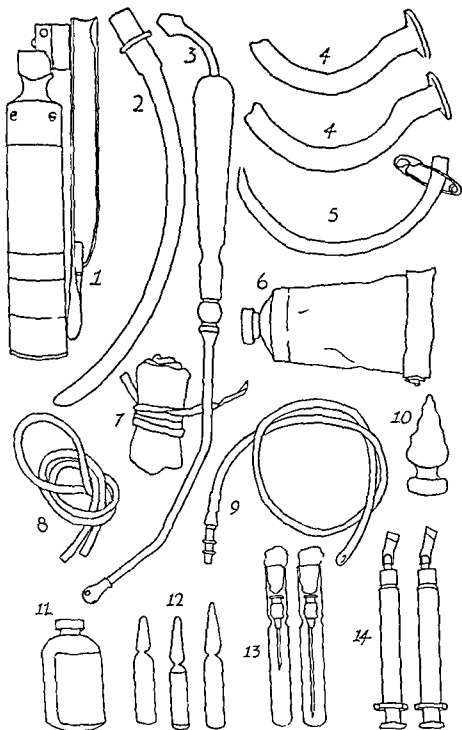


Fig 46 Contents of E Kit 1 Laryngoscope 2 Endotracheal tube 3 Metal suction tip 4 Oropharyngeal airway 5 Nasopharyngeal airway 6 Lubricant 7 Bite block 8 Tourniquet 9 Suction catheter 10 Jaw-opener 11 Short acting barbiturate 12 Ampules containing pressor drugs 13 Needles 14 Syringes

should be turned from side to side hourly to minimize hypostatic pulmonary congestion

Support of the Circulation

An intravenous infusion of 5 per cent glucose in water should be started. Twenty-five hundred milliliters of glucose and 500 ml of normal saline should be given every twenty-four hours by vein. If shock is present, plasma expanders or whole blood may be indicated. When necessary, the blood pressure should be maintained within the physiologic limits for the patient by the use of vasopressor drugs. The continuous infusion of phenylephrine (Neo-synephrine) (10 mg in 1000 ml 5 per cent glucose in water) is advisable if a single intramuscular injection of 2 to 3 mg of the same drug, or appropriate dose of other vasopressor drug, has not elevated the blood pressure. If a more potent vasopressor is required, norepinephrine (4 mg per 1000 ml 5 per cent glucose in water) is indicated. The latter drug should not be tried first because of the more evanescent effect and the greater hazard of local tissue necrosis. Both solutions require careful titration. If a patient's blood pressure cannot be maintained by the concentrations of pressor drugs listed, these should be increased three- to four-fold.

DIAGNOSIS

As mentioned, once respiratory and circulatory function has been found adequate, one can turn to differential diagnosis.

History

Consult with the patient's physician and/or family and friends for further information. Determine if possible, the type and amount of drug or drugs involved. The following questions may be asked: is the individual a habitual user of drugs, or, was there concomitant use of alcohol? Does the patient have a history of cardiovascular, cerebral, hepatic or kidney disease? What treatment for poisoning has the patient had prior to admission? Caution: do not be led astray by the demonstration of empty drug bottles. The implication may not always be correct, and more than one drug may be involved.

Chemical Analysis

Barbiturates may be detected chemically in gastric contents, urine or blood. There are also methods for determining certain of the opiates. Failure of detection, however, should not exclude the fact that the drugs may be present.

Suggestive Clinical Findings

Rapid, shallow respirations are more likely to follow ingestion of a barbiturate, while slow, deep breathing is indicative of narcotic poisoning. Pinpoint pupils suggest but are not pathognomonic of narcotic overdosage. Hypoxia dilates pupils.

EVALUATION OF THE DEGREE OF CENTRAL NERVOUS SYSTEM DEPRESSION

Some attempt must be made to assess the depth of depression. A prognosis cannot be offered until such an assessment has been made. The following tests have proved of use in determining the degree of narcosis.

Respiratory response to the inhalation of 5 per cent to 10 per cent carbon dioxide. Failure of hyperpnea to appear indicates depression of the respiratory center.

Blood barbiturate levels. Levels above 10 mg per 100 ml suggest deep narcosis. Habitual users of barbiturates may tolerate higher levels than those unaccustomed to their use.

The tracheal carinal reflex. This is one of the last reflexes of the body to be abolished. Its presence or absence is determined with catheter stimulation during tracheobronchial aspiration. Its absence indicates deep coma.

Electroencephalographic tracings. Predominance of waves of low frequency and low amplitude indicates deep depression.

Response to the rapid intravenous injection of 5 ml of 10 per cent pentylenetetrazol (Metrazol). No response suggests deep depression while an increase in respiration or movement of limbs indicates a mild depression.

Absence of tendon reflexes or pupillary response to light is an indication of deep coma.

FURTHER TREATMENT IN ORDER OF IMPORTANCE

Gastric Lavage

A considerable quantity of unabsorbed drug may remain in the stomach for many hours. This can be removed by passage of a large bore gastric tube with careful aspiration of the gastric contents using 10 to 20 ml amounts of normal saline. Regurgitation of stomach contents must be anticipated. In deep coma it is best to prevent pulmonary aspiration of gastric contents by passage of a cuffed endotracheal tube before gastric lavage. After lavage, the cuffed tube can be replaced with a plain one to minimize tracheal irritation.

Prevention of Urinary Bladder Distention

This may be obviated by the insertion of an indwelling catheter or by intermittent catheterization

Prevention of Infection

Comatose individuals are prone to develop pulmonary infections. Antibiotics should therefore be used prophylactically.

Prevention of Damage to Eyes, Lips and Mouth

Care should be exercised to prevent such damage by taping the eyes shut and by lubricating the lips to prevent drying and cracking. Attention must be paid to oral hygiene.

Use of Analeptic Drugs

The value of these substances is controversial. The consensus is that they should be employed only after attention has been paid to the previous measures and only in those patients thought to be in deep coma. Their use in patients who have experienced severe hypoxia is contraindicated. The use of analeptics is associated with certain hazards. These include convulsions with subsequent cerebral depression, vomiting with the danger of aspiration of gastric contents and the production of cardiac arrhythmias.

Picrotoxin This is the most potent and most dangerous of the analeptics. The suggested dose is 3 mg given intravenously followed by a fifteen to twenty minute wait for improvement in respiration or tendon reflexes. If there is no response, a second dose of 6 to 9 mg can be administered followed by the same waiting period. The dose can be increased gradually until the following signs of improvement are noted: increase in muscular tone, improved respiratory activity and returning reflexes. Muscular twitching suggests impending convulsions. Under these conditions picrotoxin should be withheld.

Pentylentetrazol 10 per cent Solution (Metrazol) This drug is less potent than picrotoxin. The maximal action occurs within one to two minutes and the duration of action is brief. Five to 10 ml should be administered intravenously as an initial dose. This is increased and repeated as indicated.

Methyl-ethyl-glutarimide (Megimide) The efficacy and comparative potency of this drug have not yet been established. Once thought to be a specific antagonist to barbiturates, it is now recognized as a non-specific stimulant similar in action to picrotoxin. Rapidity of action

parallels that of pentylenetetrazol. The recommended dose is 10 ml of an 0.5 per cent solution given intravenously. The dose is repeated as necessary.

Nalorphine (Nalline) and Levallorphan (Lorfan) These are specific antagonists to the narcotics (Fig. 47) and are of no value, even possibly harmful, when used in patients depressed by other drugs. Levallorphan is about five times more potent than nalorphine. The intravenous dose

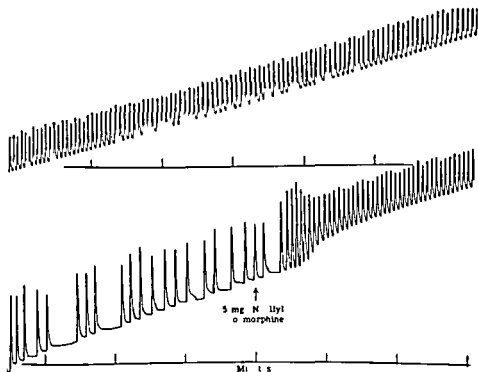


Fig. 47 Increase in respiratory rate and minute volume following the intravenous injection of nalorphine in a patient depressed by morphine (Eckenhoff, J. E., Elder, J. D. and King, B. D. *Am. J. Med. Sc.* vol. 223, 1952)

of levallorphan is 1 to 2 mg. and that of nalorphine 5 to 10 mg. Either drug may be repeated within two hours if needed.

Hemodialysis

This is a relatively new form of treatment for barbiturate poisoning and has not yet been thoroughly evaluated. By the use of an "artificial kidney," a significant proportion of barbiturate can be removed from the blood stream. Dialysis appears to be most effective for patients who have ingested overwhelming amounts of drug. There is a risk in the use of hemodialysis so that it is not advocated for any but seriously poisoned patients.

PROGNOSIS

Deep coma does not always imply a poor prognosis. However, hypoxia as a prominent feature of coma worsens the prognosis and increases the likelihood of residual cerebral damage. The presence of concomitant cardiovascular, cerebral or hepatic disease bespeaks a poorer prognosis. Absence of the tracheal carinal reflex carries with it a poor prognosis, as do electroencephalographic tracings indicative of persistent deep narcosis. Combinations of drugs such as barbiturates and alcohol appear to cause death more often than if alcohol has not been taken.

APPRAISAL

The available data suggest that the mortality from barbiturate poisoning treated by supportive therapy alone can be strikingly small. It is probable that analeptics do not increase the survival rate and may even increase mortality. We therefore rarely use this type of treatment. Comatose patients require vigilant attention and should never be left alone. Observations should be recorded carefully by nurses as well as physicians. With such care and application of the aforementioned therapy, the mortality and morbidity from narcotic poisoning should be minimal.

REFERENCES

- Berman L B, Jeghers H J, Schreiner G E and Pallotta A J. Hemodialysis, an effective therapy for acute barbiturate poisoning. *J A M A*, 161:820, 1956.
Eckenhoff J E, and Dam, W. The treatment of barbiturate poisoning with or without analeptics. *Am J Med*, 20:6, 1956.
Fisher, R. S., Walker J T, and Plummer C W. Quantitative estimation of barbiturates in blood by ultra spectrophotometry. II. Experimental and clinical results. *Am J Clin Path*, 18:562, 1948.
Nilssen, E. On treatment of barbiturate poisoning. *Acta med Scandinav*, Suppl. 139, 1951.
Shulman, A., Shaw, F H, Cass, N M and Whyte H M. A new treatment of barbiturate intoxication. *Brit M J*, 1:1238, 1955.

Fundamentals of Inhalation Therapy

DURING the early decades of the twentieth century, hospitals maintained oxygen tents but little interest in the rational use of oxygen was displayed by either the medical or administrative staffs. No attempt was made to estimate the concentration of oxygen inside the tent, and the majority of patients breathed a far lower concentration of oxygen than their medical attendants realized. Measurements of arterial oxygen content, saturation or tension were confined to research laboratories.

Gradually more diversified methods for increasing the alveolar oxygen tension were developed and nasal catheters, masks and hoods came into clinical use. Yet the indications for a particular device remained poorly understood by most physicians, as did the operational aspects of the equipment involved. The nursing and orderly services were usually in charge of setting up and supervising the apparatus selected. At this stage of development the term oxygen therapy was coined. It connoted an effort—usually not verified by objective tests—to increase the amount of oxygen available to a patient's lungs.

Attention began to turn toward the respiratory tract as a whole. Physician specialists in anesthesia emphasized the problems in pulmonary gas exchange associated with improper positioning of the unconscious patient, the encroachment of tumors upon the airway, and the accumulation of secretions within the respiratory tract. Anesthetists were consulted when respiratory problems developed in patients on all hospital services, and a subdivision of oxygen therapy was often established within a department of anesthesia. Anesthetists understood respiratory tract obstruction, they were familiar with the management

PROGNOSIS

Deep coma does not always imply a poor prognosis. However, hypoxia as a prominent feature of coma worsens the prognosis and increases the likelihood of residual cerebral damage. The presence of concomitant cardiovascular, cerebral or hepatic disease bespeaks a poorer prognosis. Absence of the tracheal carinal reflex carries with it a poor prognosis, as do electroencephalographic tracings indicative of persistent deep narcosis. Combinations of drugs such as barbiturates and alcohol appear to cause death more often than if alcohol has not been taken.

APPRAISAL

The available data suggest that the mortality from barbiturate poisoning treated by supportive therapy alone can be strikingly small. It is probable that analeptics do not increase the survival rate and may even increase mortality. We therefore rarely use this type of treatment. Comatose patients require vigilant attention and should never be left alone. Observations should be recorded carefully by nurses as well as physicians. With such care and application of the aforementioned therapy, the mortality and morbidity from narcotic poisoning should be minimal.

REFERENCES

- Berman L. B., Jeghers, H. J., Schreiner G. E. and Pallotta A. J. Hemodialysis, an effective therapy for acute barbiturate poisoning. *JAMA* 161 820 1956.
Eckenhoff J. E. and Dam W. The treatment of barbiturate poisoning with or without analeptics. *Am J Med*, 20 6 1956.
Fisher, R. S., Walker J. T., and Plummer, C. W. Quantitative estimation of barbiturates in blood by ultra spectrophotometry. II. Experimental and clinical results. *Am J Clin Path* 18 562 1948.
Nilssen, E. On treatment of barbiturate poisoning. *Acta med Scandinav*, Suppl 139, 1951.
Shulman A., Shaw, F. H., Cass N. M. and Whyte H. M. A new treatment of barbiturate intoxication. *Brit M J* 1 1238 1955.

eters should be changed from nostril to nostril at least every twelve hours, for they become encrusted and the nose becomes sore

Masks

By means of face masks one can administer higher concentrations of oxygen (90 to 100 per cent). However, the disadvantages are considerable. If a good mask fit cannot be obtained, leakage will reduce the oxygen concentration available. Masks are uncomfortable for prolonged periods of time. Frequently, in restless or uncooperative patients, the mask becomes dislodged. Constant attention is mandatory. Because of the increase in dead space, carbon dioxide may accumulate. The latter factor is important in the use of the BLB mask, in which there is partial rebreathing. It is less important in the OEM or non-rebreathing mask.

Oxygen Tents or Hoods

With these devices the patient breathes from a larger volume than with a mask. Leaks must be prevented and the oxygen flow into the tent or hood must be sufficient to prevent accumulation of carbon dioxide, that is, 15 liters per minute for 20 to 30 minutes when the patient first is placed in the tent and 10 to 12 liters per minute thereafter. The oxygen content of a tent is rarely above 50 per cent, and may be little more than that in room air unless care is exercised to prevent leakage. The oxygen content of a hood can reach 90 to 100 per cent with care. When either of these techniques is used, the oxygen concentration should be measured frequently by drawing samples from the hood or tent through an oxygen analyzer. The oxygen content may fall below normal in a tightly closed tent or hood if the supply is exhausted. Constant attention is therefore required.

POSSIBILITIES OF HARM FROM OXYGEN

Treatment of Chronic Hypoxia

The administration of high oxygen concentrations to patients with chronic hypoxia may result in coma. The physiologic basis for this is probably as follows. First, it has been shown that strong chemoreceptor stimulation may result in stimulation of the cerebral cortex as well as of the medullary centers. A low tension of oxygen in the arterial blood is a potent stimulant to the chemoreceptors of the carotid and aortic bodies. If hypoxia has persisted for a long period, its sudden removal by inhalation of oxygen might lead to a depression of cerebral cortical cells and appearance of coma. In addition, the patient with pulmonary

of gases under pressure and they were skilled at direct laryngoscopy. The subdivision was therefore a natural one.

As knowledge of pulmonary function accumulated and the pathologic physiology of such respiratory diseases as pulmonary emphysema, fibrosis and asthma became better understood, the field broadened. The importance of factors other than the administration of oxygen and the maintenance of a patent airway was realized. The value of humidification of inspired gases and, the use of aerosols and bronchodilator drugs was explored. The basic aim of this interest was to oxygenate blood, to remove carbon dioxide from the alveoli and to diminish the respiratory effort required to accomplish these purposes. This broader approach is now known as inhalation therapy, the fundamentals of which will be presented in this chapter.

INCREASING THE OXYGEN CONTENT OF INSPIRED AIR

If the passage of gas into the alveoli is unimpeded and the oxygen content of arterial blood is below normal as evidenced by cyanosis or blood analysis, the inspired oxygen concentration should be increased. This can be accomplished by the following techniques:

Nasopharyngeal Catheter

A soft rubber or plastic catheter lubricated with a water-soluble jelly is passed through the nose and the tip placed just above the epiglottis. The proper distance can be estimated for each patient by placement of the catheter along the patient's face with the tip of the catheter at the tragus of the ear. The distance from tragus to the external nares minus 1 cm. is the approximate length of catheter to be inserted. In conscious patients, the proper distance can be estimated by inserting the catheter with oxygen flowing at 6 liters per minute, until the patient begins to swallow the delivered oxygen. The catheter should then be withdrawn about 1 cm. and fastened to the forehead and nose, or to the upper lip and lower cheek. If it is advanced too far, the patient will swallow oxygen and the stomach may become distended. If it is not advanced far enough, the concentration of oxygen inspired will be too low. Properly placed, and with a flow of 6 liters per minute, an oxygen concentration of about 50 to 55 per cent can be maintained in the air entering the trachea. The oxygen should be humidified by passage through water to prevent drying of the mucous membranes. The catheter may also be placed within an endotracheal or tracheostomy tube, taking care not to occlude these airways. The flow of oxygen in these latter circumstances need not exceed 4 liters per minute. Cath-

Consequently oxygen apnea is limited to a small group of patients, such as the following (1) Patients deeply anesthetized with non-volatile anesthetics, such as barbiturates, Avertin and opiates, which depress the normal response of the center to carbon dioxide, and so depress respiration and produce hypoxia. These agents do not set up reflexes stimulant to respiration (other than hypoxia) as does ether (see page 57) (2) Patients with cerebral trauma or edema with consequent damage to the respiratory center (3) Normal individuals made severely hypoxic by exposure to low oxygen mixtures or to high altitudes. Under these circumstances medullary depression may have been caused by severe hypoxia, or the hyperventilation induced by hypoxia may have lowered the arterial carbon dioxide tension to the point at which it became ineffective in stimulating the respiratory center. The incidence of this type of apnea is low, the duration brief, and no special measures are needed to start breathing again.

The occurrence of 'oxygen apnea' does not mean that inhalation of oxygen should be stopped. It is merely a convincing indication of the need for such therapy, even if artificial ventilation is required temporarily. It is far better to have an individual well oxygenated through artificial respiration than to have him breathing spontaneously and be poorly oxygenated.

Oxygen Toxicity

If oxygen is inhaled for longer than 24 hours and the inspired concentration is greater than 70 per cent, one must anticipate the possibility of oxygen toxicity. In normal man and animals, the inhalation of high concentrations of oxygen for this length of time produces signs and symptoms of respiratory tract irritation. These consist of substernal pain, cough, dyspnea, and occasionally pneumonitis. The cause of this syndrome is unknown. Whether a longer or shorter time would be required for its development in patients with respiratory tract disease is also not known.

ADJUNCTIVE THERAPY

Humidified Gas

In the presence of high flows of gas with low moisture content, the respiratory mucous membranes become dry, ciliary activity is reduced, secretions thicken and are difficult to expel. The air passages narrow or become blocked and decreased alveolar ventilation and atelectasis develop. This sequence of events can be minimized by the inhalation of gas with a high moisture content. Means for providing humidified gas

emphysema usually has difficulty with the pulmonary exchange of both carbon dioxide and oxygen. As a result, a prolonged elevation of the arterial carbon dioxide tension occurs, and the respiratory center may become insensitive to carbon dioxide. The patient's respiration is then driven partly by hypoxia and partly by vagal reflexes from the lung but not by carbon dioxide. Under these circumstances inhalation of high concentrations of oxygen decreases tidal exchange by removal of part of the reflex drive to the respiratory center. Carbon dioxide may then accumulate to toxic, narcotic levels and so further depress the cerebral cortex.

Serious depression of the central nervous system by oxygen occurs in only a small percentage of patients with chronic hypoxia. However, when it does occur, the physician faces a dilemma. Untreated, the patient may be conscious but owing to the hypoxia, cerebral and cardiac damage may result. Treated with oxygen, the patient's arterial blood becomes better saturated but coma ensues.

Oxygen Apnea

A deeply narcotized patient may stop breathing following a few breaths of oxygen. This has been termed "oxygen apnea." The following explanation has been offered for this phenomenon. In a normal individual, respiration is regulated primarily by carbon dioxide acting directly upon the medullary respiratory center, and to a lesser degree, by reflexes arising within the vascular system and in the airway and lungs. In the narcotized patient these reflex mechanisms become more important in controlling respiration because all cerebral depressants reduce the sensitivity of the medullary centers to carbon dioxide. In deep anesthesia the response to carbon dioxide may be completely lacking and the only forces maintaining respiration may be those arising from the more resistant reflex mechanisms such as the oxygen sensitive chemoreceptors. In such a condition, an abrupt correction of the hypoxia may result in temporary apnea.

The important question from the clinical point of view is "When may such apnea be expected to occur?" It may be anticipated when oxygen is given to patients whose respiratory center is no longer regulated normally by carbon dioxide and who have no important reflexes, other than chemoreceptor reflexes, maintaining respiration. These qualifications exclude the majority of patients with anoxia due to pathologic changes in the lungs, for the respiratory center in such individuals is being stimulated by another important reflex, the vagal stretch reflex (Hering-Breuer).

ance to breathing when there is turbulent flow because the density of the gas mixture is low. It probably will not change resistance to respiration when turbulence is not a factor since the viscosity of a helium-oxygen mixture is slightly greater than that of air.

Oxygen under Increased Pressure

Ventilation occasionally must be assisted by raising the pressure in the air passages above atmospheric. When this situation arises, there is usually suboxygenation of arterial blood so that in addition to the increased pressure, oxygen should be added to the inspired mixture. Several illustrations of the utility of this therapy will be presented. In pulmonary edema the lungs become inelastic and do not respond as readily to normal changes in intrapleural pressure. The air passages contain frothy fluid. Under these circumstances increased airway pressure may expand the lungs more adequately and provide better alveolar ventilation. The additional oxygen raises the oxygen tension within the alveoli, thus increasing the diffusion gradient across the pulmonary capillary membrane, but the mechanical dilatation provided by the added pressure is probably of greater importance. Similarly in pulmonary atelectasis, collapsed alveoli can be expanded by increased airway pressure. Again, the mechanical dilator or expansion effect is probably of as much value as the additional oxygen.

Oxygen can be administered under increased pressure by a bag and mask for short periods, but a mechanical ventilator is more satisfactory for prolonged treatment. The ventilator of choice is one activated by the patient's breathing and in which both expiratory and inspiratory pressures can be varied by the attendant. It is not feasible in this chapter to discuss the features of each of the many devices available. The student must become familiar with the mechanical aspects and limits of applicability of the equipment which he is to use. Only in this way can he diagnose and correct inefficient or improper operation.

Carbon Dioxide Inhalation

A rise in the carbon dioxide tension in arterial blood will increase the rate and depth of respiration unless the respiratory center is depressed by narcotics or disease, the muscles of respiration cannot respond normally to this stimulus as in anterior poliomyelitis, or the lungs and chest wall are stiffened and fixed as in pulmonary emphysema. Carbon dioxide inhalations may be used in the prevention and treatment of pulmonary atelectasis, in the liquefaction of respiratory

include the use of cold steam, nebulization of water in a croup tent or standard oxygen tent, or inhalation directly from a nebulizer. In addition to water, one can nebulize a wetting agent such as propylene glycol or commercial preparations such as Alevaire. The inhalation of humidified gases is of value in the treatment of acute laryngotracheobronchitis, of chronic bronchitis superimposed upon pulmonary emphysema, or when bronchiolitis complicates bronchial asthma.

Certain cardiovascular stimulants such as epinephrine, isoproterenol (Isuprel) and phenylephrine (Neo-synephrine) can dilate bronchioles by reducing smooth muscle tone and decreasing mucous membrane congestion through vasoconstriction. This effect can be achieved by systemic administration of the drugs but topical application is preferable to minimize undesirable side actions. Nebulization is the term used to describe the formation of microscopic particles of fluid. These can be inhaled and because of their small size will reach the smallest air passages. To decrease the deposition of the drugs upon the walls of larger conducting tubes, they should be dissolved in a vehicle such as 10 to 15 per cent propylene glycol, the combination appearing to penetrate deeper into the lungs than when water alone is used as the diluent. The patient inhales the nebulized vapor for 8 to 10 minutes several times a day. Secretions become more easily expelled, the air passages are cleared and dilated and the flow of gas in and out of the alveoli is made easier. This form of treatment must be used with caution in patients with congestive heart failure, evidence of myocardial ischemia, and hypertension, since some of the inhaled drug reaches the systemic circulation.

Dilution of Oxygen with Helium

As Comroe and his associates have pointed out, airway resistance depends in part upon the nature of airflow—which may be laminar (streamlined) or turbulent. The pressure required to produce laminar flow is proportional to the volume flow times a constant, which is related to the viscosity of the gas; it is independent of the density of the gas. The pressure required for turbulent flow is proportional to the square of the volume flow times another constant related to the density of the gas; it is independent of the viscosity of the gas. The tracheo-bronchial tree has hundreds of thousands of branchings, and eddy formations may be set up at these. Turbulence or eddy formation is particularly apt to occur when there are irregularities in the tubes, such as might be caused by mucus, exudate, tumor or foreign bodies. Inhalation of 80 per cent helium and 20 per cent oxygen will decrease resist-

Death Reports

THE CONSCIENTIOUS anesthetist will carefully analyze the circumstances surrounding the death of every patient who has received an anesthetic. After deliberation a decision should be reached as to whether a written summary of the events should be prepared. If the anesthetic management, viewed in its broadest aspects, appears to have had no influence on the fatality, a report is not written. If anesthesia is judged to be a major contributing factor, a detailed analysis is indicated. In some instances a cause-effect relationship between anesthesia and death is less certain, but suggestive. Examples of this include the following:

- 1 A patient retches violently during the postoperative period, ruptures the abdominal incision and dies as the result of this complication.
- 2 Severe hypotension occurs during operation and a fatal cerebral or coronary thrombosis occurs 24 to 36 hours later.
- 3 Liver failure follows prolonged hypotension during or after an operation.
- 4 A fatal thyroid crisis follows hypoxia related to respiratory obstruction during anesthesia.
- 5 A delayed death from pneumonia or lung abscess results from aspiration during or following anesthesia.

These cases deserve just as careful analysis as those in which death can be directly attributed to anesthesia. The careful preparation of a report is of great educational value to the individual who administered the anesthetic. Discussion of these reports at a conference of anesthetists or by an Anesthesia Study Commission is also enlightening. In the aggregate such information can be used to assess the many factors which contribute to a fatal outcome. We believe that death reports are

tract secretions, and in the treatment of carbon monoxide poisoning and hiccoughs

A variety of methods of administering carbon dioxide may be used. One can rebreathe from a paper bag, inhale 5 per cent or 10 per cent carbon dioxide in 95 per cent or 90 per cent oxygen from a bag and mask, or breathe 100 per cent carbon dioxide supplied by the gravity method. With the latter technique the delivery tube from the carbon dioxide tank is held three to four inches from the patient's nose or mouth and the carbon dioxide allowed to flow over the face. If hyperpnea does not appear within 30 seconds by the gravity method, it should be discontinued. With the other methods, two to three minutes should suffice.

ANESTHESIA AND INHALATION THERAPY

Most anesthetists do not have the time to properly direct an inhalation therapy unit. Both services have grown in complexity and demand to the point that each requires full-time supervision. We believe that members of the anesthesia staff should act as consultants in clinical problems pertaining to respiratory function, but should not assume the burdens of care and distribution of equipment, training of personnel, and day and night attention to patients. These are essential to the effective operation of an inhalation therapy service. This chapter has outlined the scope of knowledge which might be expected of such a consultant. It has not been concerned with the details of creating and maintaining the service itself.

REFERENCES

- Barach, A. L. *Physiologic Therapy in Respiratory Diseases*. J. B. Lippincott Co. Philadelphia, 1948.
- Comroe, J. H. and Dripps, R. D. *The Physiological Basis for Oxygen Therapy*. Charles C. Thomas, Springfield, Ill., 1950.
- Comroe, J. H., Forster, R. I., Dubois, A. B., Briscoe, W. A. and Carlson, E. *The Lung*. Year Book Publishers, Chicago, 1955.
- Saklad, M. *Inhalational Therapy and Resuscitation*. Charles C. Thomas, Springfield, Ill., 1953.

hours of death and discussed with the head or acting head of the department within 48 hours of death

SHORT FORM

- 1 Name last, first Race, sex, age, physical status
- 2 Date of admission
- 3 Date of last anesthesia
- 4 Date of death
- 5 Hospital history number
- 6 Anesthetic agents
- 7 Past history, including previous anesthetics
- 8 Present illness, physical examination and laboratory findings
- 9 Surgical diagnosis and proposed operation
- 10 Anesthesia—complete description including premedication, time given, effect, technique, course, complications and recovery
- 11 Operation—surgeon, surgical procedures, surgical complications Anesthetist's opinion of the degree of surgical trauma
- 12 Postoperative course and treatments
- 13 Death—date, time after operation, description
- 14 Clinical cause of death
- 15 Pathologic findings
- 16 Comments

an essential feature of the teaching and clinical practice of anesthesia. An anesthetist or a Department of Anesthesia not making such analyses overlooks an opportunity for self-appraisal and self-improvement.

We suggest two forms, one longer than the other, but both providing useful information.

LONG FORM

- 1 Name last, first Race, sex, age, physical status
- 2 Date of admission
- 3 Date of last anesthesia
- 4 Date of death
- 5 Hospital history number
- 6 Anesthetic agents
- 7 Family and past history, including previous anesthetics
- 8 Present illness, physical examination and laboratory findings
Course in hospital prior to operation
- 9 Surgical diagnosis and proposed operation
- 10 Anesthesia—complete description including premedication, time given, effect, technique, course, complications and recovery
- 11 Operation—surgeon, surgical procedure, surgical complications
Anesthetist's opinion of the degree of surgical trauma
- 12 Recovery room record
- 13 Postoperative course and treatments, including type and amount of fluids, narcotics, oxygen, analeptics, vasopressor drugs
- 14 Death—date, time after operation, description (if during anesthesia or operation, include under 10 and 11)
- 15 Discussion of probable causes of death
- 16 Cause of death according to the anesthetist who did case
- 17 Cause of death according to supervising anesthetist
- 18 Cause of death according to head of department
- 19 Moral to be drawn
- 20 Additional notes
- 21 Autopsy findings, autopsy number
- 22 Cause of death according to pathologist
- 23 Pathology conference—date, decision
- 24 Anesthesia Study Commission—date, decision
- 25 Final decision on causes of death (anesthesia, condition of patient, operation etc.)
- 26 Condition initiating events leading to death

Report on items 10, 11, 12, 13 and 14 should be written within 24

Pre-clinical Years

In conjunction with anatomy and physiology a clinic on artificial ventilation may be held, correlating the property of the elastic recoil of the lungs, the role of intrapleural pressure, and other aspects of respiratory physiology with problems of respiratory obstruction and depression. Another clinic on the applied anatomy of regional anesthesia is worth-while to illustrate the value of topical landmarks as well as knowledge of structures beneath the skin. Anesthesia of the brachial or lumbosacral plexus and lumbar puncture are appropriate topics. Clinics may be given on the applied pharmacology of the drugs used in anesthesia. Anesthetic drugs and adjuncts are among the most spectacular in their action. This concept is in line with current ideas of correlated teaching of basic science and clinical medicine.

Clinical Years

Seminars or lectures may be offered during the course in obstetrics on infant resuscitation, and during the course in internal medicine on the management of patients with narcotic poisoning, agitation or tetanus. Correlation clinics may be given with surgeons on such topics as shock, hemorrhage and trauma, or with neurosurgeons, neurologists and radiologists, on the mechanisms and treatment of pain. Lectures and demonstrations on clinical anesthesia might embrace the following topics. The physician's responsibility to the unconscious patient, drugs used for preanesthetic medication, principles of inhalation, intravenous, spinal and local anesthesia, problems posed by pediatric and geriatric patients, and the anesthetic management of patients with diseases of the heart, lungs, liver, kidneys and endocrine glands. In these lectures items of broad interest to the practitioner of medicine should be stressed, that is, the protean actions of the opiates, the hazards of lumbar puncture, the consequences of hypoxia and hypotension.

Practical instruction in clinical anesthesia should be offered. We believe that among his professional skills a physician should have the following abilities: (1) to administer an anesthetic and to supervise a patient under the influence of that anesthetic, (2) to recognize and treat obstruction, depression or cessation of respiration, and (3) to treat inadequacy of the circulation. The medical student should be taught these so that in the future he need not be helpless, unable, unwilling, or afraid to face such problems. As a by-product of these experiences the student should gain knowledge permitting him to offer better care.

Instruction in Anesthesia

WE LIVE in a thermonuclear age. Should the lethal weapons of this era be used in warfare, huge numbers of casualties would result. How could anesthesia be provided for the treatment of the injured? Nurses trained in anesthesia would be used to the limit of availability. Rapid training programs could be initiated for medical and dental students, service personnel, nurses, and for such lay groups as the Emergency Aid, Red Cross, and other volunteer agencies. These programs, directed by anesthesiologists, would have only limited objectives. In addition to resuscitative measures, these would include the recognition and treatment of respiratory obstruction and inadequate pulmonary ventilation, the treatment of low blood pressure, and an acquaintance with the signs exhibited by unconscious or anesthetized patients. This would provide individuals with a useful orientation. Some could administer anesthesia under supervision. Others might be able to assume limited responsibility once anesthesia had been induced by someone more skilled.

The threat of mass casualties and the problems it poses supply a cogent reason for teaching the basic principles of the safe practice of anesthesia. Even without this reason, there is justification for their inclusion in medical and nursing curricula. Our concept of the anesthetic training of students will be developed in this chapter. Suggestions for ancillary didactic teaching programs at various levels will also be offered.

THE MEDICAL STUDENT

A department of anesthesia can contribute to the student's education during all four years of the medical school program.

THE INTERN OR SURGICAL RESIDENT

The intern or surgical resident should be assigned to the anesthesia service for periods ranging from two to eight weeks. His should be a wider experience than that offered the medical student, yet always with adequate supervision. He should administer inhalation anesthesia in closed and open systems, with ethyl ether, diethyl ether, cyclopropane and nitrous oxide, he should give thiopental and muscle relaxants by vein, and he may use basal narcosis in the form of Avertin or thiopental by rectum. An attempt should be made to instruct him in the rationale, anatomy and technique of direct laryngoscopy and tracheal intubation for use in anesthesia, in resuscitation, and as a substitute for therapeutic bronchoscopy.

Instruction should be directed to single dose techniques for spinal anesthesia, using various drugs. He should be expected to observe and, when possible, to perform regional blocks for diagnosis, therapy or operation. These should include stellate ganglion, cervical plexus, brachial plexus, intercostal, lumbar paravertebral, inguinal and caudal nerve blocks.

In addition to the administration of anesthetics in the operating room, the intern or resident should prescribe preanesthetic medication for the patients to whom he is assigned. Like others in anesthesia, he should make pre- and postoperative rounds and keep anesthesia records.

Such a period of training for a surgical resident subsequently will enable him to appreciate problems in anesthesia and to assess these more intelligently. He will have a better understanding of the necessity and value of cooperation with his colleagues in anesthesia.

THE ANESTHETIC RESIDENT

Details of a residency training program in anesthesia have been published by the Council on Medical Education of the American Medical Association in the booklet "Essentials of an Approved Residency." We shall add only a few observations.

Formal instruction during the administration of anesthetics must be a part of residency training, just as similar instruction is a part of training in other specialties. Although a resident should be able by his own observation, practice, discussion and reading to round out his abilities as an anesthetist, he can learn the experience of others only by word of mouth. This cannot be had solely through observation or by resort to the literature. In no other way can he become fully aware of the many

to patients with such abnormalities as narcotic poisoning, diabetic coma, uremia, increased intracranial pressure, poliomyelitis, tetanus, Ludwig's angina, angioneurotic edema, bronchial asthma, cardiac failure, aspiration of vomitus or foreign bodies, asphyxia neonatorum, hemorrhage, syncope, convulsions and nerve gas poisoning. In these conditions emergencies involving the respiratory and circulatory systems must be met promptly and intelligently.

During his time on the anesthesia service, a student should administer inhalation agents with open and closed techniques and should administer intravenous anesthesia and spinal anesthesia. With these methods almost any operation can be performed satisfactorily. A knowledge of a gas machine is useful since it teaches the student to handle compressed gases and the apparatus offers a ready source of oxygen for resuscitation and artificial respiration. The anesthetic agents to be used by medical students should be limited to ethyl ether, divinyl ether, nitrous oxide, cyclopropane, intravenous barbiturates, procaine and tetracaine. Other drugs may be used but it is unwise to train students in the use of substances which are more difficult to administer safely. The reasons for failing to emphasize the use of these should be explained to the student.

The material to be stressed should include the following, most of which is not taught elsewhere in medical schools: the use and abuse of preanesthetic medication, the signs and stages of general anesthesia, the use of artificial pulmonary ventilation, the careful performance of a lumbar puncture or venipuncture, the control of the level of spinal anesthesia, and the management of circulatory collapse. One member of the anesthesia departmental staff should be assigned to medical students as his sole responsibility. In order that this assignment not become burdensome, it should be rotated weekly or monthly. Whenever possible, this staff member should hold an orientation discussion at the beginning of the teaching session, make preoperative and postoperative rounds with the students, supervise the administration of anesthesia by the students, and organize discussion groups.

Considerable tact and patience are required of both instructor and student. The patient must not know that an inexperienced individual is administering anesthesia. A thorough discussion of the planned procedure should be carried out between the student and instructor out of range of the patient's hearing. This is particularly true of instruction during spinal anesthesia. It is bad for a patient's morale to hear such remarks as "don't do that," or "you put the needle in too far."

INDEX

- ABDUCTORS nerve palsy of after spinal anesthesia 112
- Accidents during anesthesia, 173
- Acetylcholine effect of local anesthetics, 119
 - in muscle contraction, 66
- Afferent lemnisco-thalamic system 42
- Air block due to gas pressure in lungs, 169
- Air embolism during anesthesia, 170
- Air, functional residual 37
- Airway artificial 45 46
 - obstruction, 183
 - raised pressure to, effect on arterial pressure, 160
- Alcoholics, chronic anesthesia for 41
- Allergic reactions to local anesthetics, 121
- Alphaprodine, preanesthetic use 12, 13
- Ambulatory patients anesthesia for 219-221
- American Society of Anesthesiologists, classification of physical status 6
- Amniotic fluid, aspiration by infant 228
- Amytal preanesthetic use, 10
- Analeptic drugs in narcotic poisoning, 236
- Analgesia with ethyl ether 56
- Anaphylactic reaction to local anesthetics 121
- Anectine See *Succinylcholine*
- Anesthesia
 - choice of 7
 - during operation 124-177
 - epidural, 114
 - for ambulatory patients 219-221
 - general concentration of anesthetic in arterial blood 146
- Anesthesia, general, concentration of anesthetic in end expiratory gas 146
 - determination of depth, 137-148
 - electroencephalogram in 144
 - signs of 143
 - stages of, 138
- in chronic alcoholics 41
- in shock, 41
- inhalation 36-50 See also *Inhalation anesthesia*
 - instruction in, 250-254
 - anesthetic resident 253
 - intern or surgical resident, 253
 - medical student, 250
- intravenous barbiturate 84-94 See also *Barbiturates intravenous*
- local, fundamentals 116-122 See also *Local anesthesia*
- neurophysiologic basis 42
- obstetric 222-230 See also *Obstetric anesthesia*
 - open drop 61-64 See also *Open drop anesthesia*
- pediatric 211-218 See also *Children*
- induction 215
- maintenance 215
- preparation in operating room for 18
- rectal 215
- reticular activating substance and, 42
- spinal 95-115 See also *Spinal anesthesia*
 - unusual complications 168-174
- Anesthesiologist medicolegal responsibility of 176
- Anesthetic machine 21-26 See also *Machine anesthetic*
- Anesthetic resident, instruction 253

small mistakes which bar the way to a more perfect technique. These mistakes are particularly important during the formative period *when habits become fixed*, false notions become perpetuated—and when the new resident is so overwhelmed with the complexity of administering anesthesia that he is incapable of exercising suitable judgment. This does not mean that the beginner should not have the opportunity to make mistakes, but rather, having made them, he should, with the aid of his supervisor, be able to assess the mistakes and gain advantage from them. The sooner one becomes proficient in the administration of anesthesia and becomes the master of rather than the slave of technical procedures, the sooner can he free his mind (and to some extent his hands) and give thought to broader matters.

A department of anesthesia (and physician anesthesia as a whole) will be judged by the men trained as well as by the investigative work done. It will be judged by the failures as well as by the successful men. A good resident under a *laissez-faire* program will eventually become a proficient anesthetist. A mediocre resident will become a good anesthetist only by being taught—by being led to his goal. Since the clinical practice of anesthesia depends upon perfection of technique as well as judgment, the novice should adopt fundamentally sound practice from the beginning. Such a plan should lead to safer anesthesia.

Theoretical knowledge and technical skill avail little if an individual cannot exert control of his emotions and activities under stress. The physician whose mental processes “freeze” in an emergency may be dangerous. Surgeons are quick to sense emotional tenseness or uncertainty on the part of an anesthetist. Confidence is thereby destroyed, and the anesthetist’s effectiveness lessened. One should attempt self-control at all times so that clear, logical thinking can be exercised.

- Brain action of anesthesia upon 42
 Bromo-chloro trifluoroethane 59
 Bronchospasm 8, 12 89 171
- CALCIUM chloride in cardiac arrest 207
 Carbon dioxide absorbers 24, 25
 Carbon dioxide inhalation therapy, 245
 Carbon dioxide, effect on electroencephalogram 145
 properties 28
 respiratory response to with narcotics 11
 with cyclopropane, 54
 Cardiac arrest
 anesthetist's role in, 208
 diagnosis, 205
 factors in, 204
 prognosis 210
 treatment 206
 Cardiac resuscitation 204-210
 Cardiac rhythm cyclopropane and 54
 Cardiovascular stimulants nebulized 244
 Catheter nasopharyngeal for oxygen therapy 240
 Catheterization in narcotic poisoning 236
 Caudal anesthesia continuous for normal vaginal delivery 226
 Cautery equipment safety factors in use of 153
 Central nervous system damage to
 from cardiac arrest 210
 depression in narcotic poisoning 235
 notes for anesthetic chart 134
 reactions to local anesthesia 120
 Cerebral stimulants in treatment of emergence excitement 197
 Cesarean section elective, anesthesia for 227
 emergency anesthesia 228
 Chart anesthetic See *Record anesthetic*
 Chemical sterilization of equipment 199
 Childbirth anesthesia during 222-230
 See also *Obstetric anesthesia*
 Children
 anesthesia in 211-218
 anesthetic management 212
 blood pressure in 214
 blood transfusion in 216
 convulsions due to anesthesia 218
 endotracheal anesthesia in 217
 fluid requirements 214
- Children, larynx in, 217
 outpatient operations in instruction for parents, 219
 preanesthetic medication, 211
 preparation for operation, 211
 respiratory and circulatory data, 213
 Chloral hydrate, preanesthetic use, 10
 Chloroform, anesthetic use 59
 partition coefficient 38
 Chlorpromazine
 effect on circulation, 15 156
 on course of anesthesia 157
 in treatment of postoperative excitement 197
 postural hypotension due to, 15
 preanesthetic use, 14
 Circulation
 abnormalities of, arterial hypotension and 165
 depression due to local anesthetics, 120 See also *Hypotension arterial*
 effect of narcotics on, 12, 13
 in children, 213
 support in narcotic poisoning 234
 supportive measures during anesthesia, 166
 Circulatory system, notes for anesthetic chart, 135
 Classification of physical status 6
 Cleaning of equipment for inhalation anesthesia, 198-201
 Closed system technique of anesthesia, safety factors in 151
 Cobefrin with local anesthetics 122
 Cocaine as local anesthetic 117
 Complications unusual of anesthesia, 168-174
 Continuous caudal anesthesia for normal vaginal delivery 226
 Controlled respiration apnea due to 70
 Convulsions, divinyl ether and 58
 due to anesthesia in children 218
 due to local anesthetics 120
 Cornea injury to during anesthesia, 62, 168
 Cortisone effect on course of anesthesia, 156
 Cough due to intravenous barbiturates, 92
 during inhalation anesthesia 46
 Cracking gas cylinders 21 30
 Curare See *Muscle relaxants*
 Cyclopropane 52
 effects of 52
 circulatory, 54

- Anesthetics**
 choice of 7
 elimination of, 51
 flammability of 149
 general, for ambulatory patients, 221
 overdose with 157
 inhalation 51-60
 amount required, factors influencing 39
 distribution in tissues, 38
 partition coefficients of 38
 local 117
 allergies to, 121
 for continuous caudal anesthesia, 226
 metabolism and excretion, 119
 rapid absorption causing hypotension, 158
 reactions to 120
 spinal dosages, 97
 duration of action, 97
 fate in subarachnoid space 107
 theories of action, 118
 volatile liquid See *Open drop anesthesia*
- Anesthetist surgeon relationships** 175-177
- Anoxia diffusion postoperative** 186
- Anticholinesterase drugs antagonism to muscle relaxants**, 68
- Antenyl preanesthetic use** 14
- Apnea due to intravenous barbiturates**, 93
 due to muscle relaxants 70
 endotracheal tube as cause of 70
 oxygen, 242
 treatment, 70
- Apomorphine in treatment of postoperative excitement**, 197
- Arachnoiditis chronic progressive adhesive after spinal anesthesia** 113
- Arrest, cardiac** 204 See also *Cardiac arrest*
- Arrhythmias cardiac, arterial hypotension and** 165
 cyclopropane and 54
- Arterial hypotension**, 155-167 See also *Hypotension arterial*
- Asphyxia neonatorum** 229
- Aspiration of amniotic fluid by infant** 228
 of gastric contents, treatment 184
- Atelectasis, postoperative** 135
 pulmonary during anesthesia 186
- Atropine, preanesthetic use**, 14
 treatment of hypotension with 157, 164
 use with preostigmine, 70
- Auscultation, blood pressure measurement by** 33
- Avertin rectal use in children** 215
- Ayre tube**, 130
- BAG reservoir** 23
- Banthine** See *Methantheline*
- Barbiturate poisoning, hemodialysis in** 237 See also *Narcotic poisoning*
- Barbiturates**
 in early stages of labor 223
 intravenous 84-94 See also *Intravenous barbiturate anesthesia*
 alkalinity 88
 effect on respiration 87, 88
 extravasation of 173
 fate in body, 85
 hypotensive effect, 88
 intra arterial injection 173
 parasympathetic stimulation 88
 preanesthetic use, 10
- Belladonna drugs, preanesthetic use**, 14
- Bladder, notes for anesthetic chart**, 136
- BLB mask** 241
- Blood, arterial concentration of anesthetic in** 146
- Blood flow cyclopropane and** 54
 distribution of anesthetic and, 38
 effect on amount of anesthetic required 41
 regional oxygen consumption and 40
- Blood loss as cause of postoperative hypotension** 182
- Blood pressure determination in children**, 214
 diastolic 33
 effect of body position on 34
 of cyclopropane on 52
 of ether on, 56
 measurement, 31-35
 auscultatory method 33
 dimensions of bag, 34
 errors in 33
 placement of cuff 32
 technique 31
 systolic 33
- Blood transfusions in children** 216
 reactions to 162, 183
 use in hemorrhage 162
- Brachial plexus nerves injury during anesthesia** 171

- Ether, divinyl open drop method, 63
 - partition coefficient, 38
- Ether, ethyl 56
 - analgesia with, 56
 - auto-oxidation of, 151
 - blood pressure and 56
 - effects respiratory, 57
 - in normal vaginal delivery 224
 - liquid insufflation in lungs, 171
 - open drop method 63
 - partition coefficient 38
 - synergism with *d* tubocurarine, 58
- Ether trifluorethyl vinyl, 59
- Ether ulceration of cornea 169
- Ethinamate, preanesthetic use, 10
- Ethyl chloride 59
- Ethylene, 56
 - partition coefficient, 38
 - properties 28
- Ethyl ether See *Ether, ethyl*
- Excitement
 - emergence 187 195
 - causes 195
 - prophylaxis 196
 - treatment 196
- Explosion hazard with anesthesia 149-154
- Extubation tracheal 79 184 217
- Eyes injury during anesthesia 168

- FACE mask fitting of, 20 44
 - for oxygen therapy, 241
- Facial nerve injury during anesthesia 172
- Fibrillation, ventricular treatment of, 208
- Fire hazard with anesthesia 149-154
- Flammability of anesthetics 149
- Flaxedil See *Gallamine*
- Flow meters 23
- Fluid requirements in children 214
- Fluid therapy for arterial hypotension 166
- Fluoromar 59
- Fluothane 59
- Functional residual air, 37

- GALLAMINE 66
 - action 67
 - dosage 66
 - metabolism and excretion, 67
- Gamphen, use in chemical sterilization, 199
- Gas cylinders, features of, 29
 - storage of, 151
- Gas, end-expiratory, concentration of anesthetic in, 146
- Gas flow, laminar, 244
 - turbulent 244
- Gas, humidified, treatment by, 243
- Gas pressure excessive injury to lungs by, 169
- Gases
 - anesthetic, supplying of, 21
 - compressed, 27-30
 - properties, 28
 - safety measures for, 27
 - steps in use of 29 30
- Gastric lavage in narcotic poisoning 235
- Gastrointestinal tract notes for anesthetic chart 136
- General anesthesia See *Anesthesia general*

- HAMMAN'S sign, 169
- Hazards of immediate postoperative period, 180-187
- Headache description of 134
 - following spinal anesthesia 107
 - treatment of 111
- Hearing difficulties after spinal anesthesia 111
- Heart See *Cardiac*
- Heat, sterilization by, 200
- Helium in inhalation therapy, 244
- Helium, properties, 28
- Hemodialysis in barbiturate poisoning 237
- Hemorrhage effect on arterial hypotension 161
 - intractable oozing 183
- Hexachlorophene, 199
- Histamine release with muscle relaxants 71
 - with narcotics 12, 161
- Hospital record preanesthetic notes for 4
- Humidified gas treatment by 243
- Hypotension
 - arterial abnormalities of circulation and 165
 - as immediate postoperative hazard 180
 - causes 155

- Cyclopropane effects of, respiratory, 52
 in cesarean section 227
 partition coefficient, 38
 pitocin and, 52
 preanesthetic medication and 52
 properties 28
 respiratory acidosis and 53
 Cyclopropane shock 182
 Cylinders gas cracking 21 30
 features of, 29
 handling of 21
 steps in use of, 29
- DAY of anesthesia 18-122
 Death reports, 247-249
 long form, 248
 short form 249
 Decamethonium
 action 66
 dosage 66
 metabolism and excretion 67
 Defibrillation electrical 208
 Delirium emergence See *Excitement emergence*
 Delivery anesthesia in 224 See also
Obstetric anesthesia
 Demerol See *Meperidine*
 Depolarization of muscle 66
 Depth of anesthesia determination of
 137-148
 Deterioration of equipment for inhalation
 anesthesia 198
 Dextran, in treatment of hemorrhage,
 162
 Dial, for chemical sterilization of equip-
 ment, 199
 Diathermy equipment safety factors in
 use of 153
 Dibucaine
 as local anesthetic 118
 spinal dosage 97
 duration of action 97
 Diffusion anoxia due to nitrous oxide
 56
 postoperative 186
 Diffusion pulmonary inhalational an-
 esthetics and 37
 Dimethyl d tubocurarine
 action 66
 dosage 66
 metabolism and excretion 67
 Disinfectants for use against tubercle
 bacillus 201
 Divinyl ether See *Ether divinyl*
- Dressings restrictive respiratory de-
 pression due to 187
 Drugs, preanesthetic, 9-16 See also
Preanesthetic medication
 Dura needle puncture openings in 108
- EDEMA laryngeal in children 217 229
 Edrophonium antagonism to muscle re-
 laxants, 68
 Electrical activity of nerves effect of
 local anesthetics 118
 Electroencephalogram, after cardiac re-
 suscitation 210
 effect of carbon dioxide on 145
 in determining depth of anesthesia
 144
 in narcotic poisoning 235
 Embolism
 air, during anesthesia 170
 arterial hypotension and 165
 pulmonary, as cause of postoperative
 hypotension 183
 Emergence excitement 187, 195
 Emergency kit, 232, 233
 Emphysema pulmonary interstitial due
 to gas pressure 169 170
 Endobronchial intubation 73
 Endotracheal anesthesia in children 217
 Endotracheal tube
 apnea due to, 70
 cuffed 82
 in asphyxia neonatorum 229
 removal problems of 184
 types of 80
 See also *Intubation tracheal*
 Ephedrine dosage 167
 effect on arterial hypotension 159
 Epidural anesthesia 114
 circulatory effects 159
 Epinephrine as cause of cyclopropane
 arrhythmias 54
 Epinephrine
 in cardiac arrest 207
 in local anesthesia 122
 in spinal anesthesia 98
 in treatment of arterial hypotension
 dosage 167
 liberation with ether anesthesia 56
 relation to emergence excitement 196
 Equipment endotracheal 81
 operating room safety factors in 152
 storage of 151 200
 Ether, divinyl 58
 convulsions with 58

INDEX

- Intubation tracheal 72-83
 appraisal 83
 blind nasal 78
 equipment, 80
 extubation in 79
 in asphyxia neonatorum 229
 in narcotic poisoning 232
 lubricants 82
 nasal blind 78
 oral in conscious patient 78
 visual curved blade 77
 straight blade 76
 postoperative complications 74
 technical complications 72
 techniques 74
 traumatic complications 72
 types of tubes 80
 undesirable effects 73
- Ischemia cerebral from cardiac arrest, 210
- Isopropylarterenol in cardiac arrest, 207
 in inhalational therapy 244
- Isuprel See *Isopropylarterenol*
- KEPHRINE with local anesthetics 122
- Kidney artificial in barbiturate poisoning 237
 notes for anesthetic chart, 136
- Kreiselman resuscitator 232
- LABOR early phases analgesia and amnesia during 223
- Laryngeal edema in children 217 229
- Laryngoscope choice of, 80
 use of 75 76
- Laryngospasm due to intravenous barbiturates 92
 during inhalation anesthesia 47
 treatment 185
- Larynx infant vs adult 217
- Lavage gastric in narcotic poisoning 235
- Levallorphan in infant resuscitation, 229
 in narcotic poisoning 237
- Levophed See *Norepinephrine*
- Lidocaine as local anesthetic, 118
- Liver notes for anesthetic chart 136
- Local anesthesia 116-122
 advantages 116
 disadvantages 117
 drugs 117
 effect of electrical activity on nerves 118
- Local anesthesia, for ambulatory patients, 220
 humoral action and 119
 reactions to 120
 central nervous system 120
 circulatory depression 120
 respiration of nerve fibers and, 119
 sensitivities and allergic phenomena, 121
 technique, 121
 theories of action 118
- Local anesthetics See *Anesthetics local*
- Lorfan See *Levallorphan*
- Lubricants 82
- Lumbar puncture technique 102
- Lungs injury to during anesthesia, 169
 excessive pressure 169
 insufflation of ether, 171
- MACHINE anesthetic elements of 21-26
- Malpractice, relation of records to 124
- Manipulation surgical arterial hypotension due to 162
- Manual systole 206
- Mask face fitting of 20 44
 for oxygen therapy, 241
- Mechanical ventilators 245
- Medical student instruction in anesthesia 250
- Medication preanesthetic guides to 9-16 See also *Preanesthetic medication*
- Medico legal responsibility in anesthesia 176
- Megimide in narcotic poisoning 236
- Membrane pulmonary diffusion of anesthetic across 37
- Meperidine
 effect on respiration 11
 in treatment of postoperative excitement 197
 postoperative use 194
 preanesthetic use 12 13
- Meters flow, 23
- Methantheline preanesthetic use 14
- Methods See *Techniques*
- Methoxamine effect on arterial hypotension 159
 in treatment of arterial hypotension 166
- Methyl ethyl glutarimide in narcotic poisoning 236
- Metrazol in narcotic poisoning 236
- Metubine See *Dimethyl d tubocurarine*

- Hypotension, arterial, changes in position or motion and 165
 due to intravenous barbiturates, 93
 during anesthesia, 155-167
 during spinal anesthesia, 106
 effect of barbiturates, 88
 of spinal and epidural anesthesia 159
 of therapeutic drugs, 156
 excessive preanesthetic medication and, 155
 hemorrhage as cause, 161
 overdose of general anesthetic and 157
 postoperative, blood loss as cause, 182
 due to change in position 181
 reduction in level of anesthesia as cause 182
 raised airway pressure and 160
 surgical manipulation and, 162
 treatment 166
 fluid therapy, 166
 pressor drugs 166
 postural due to morphine 13
 effect of chlorpromazine 15
 reflex, 163
- Hypovolemia, arterial hypotension and 165
- Hypoxia chronic, oxygen therapy in 241
 emergence excitement due to 195
 immediate postoperative 183
- ICUTIO, sources of, 151
- Induction of anesthesia in children, 215
- Infant
 blood transfusion in 216
 larynx in 217
 resuscitation of 229
 survival of dependence upon events of labor 222
 See also *Children*
- Infusion, intravenous preoperative 19
- Inhalation anesthesia agents for See *Anesthetics inhalation*
 airway and respiratory problems in 44
 amount of anesthetic required, 39
 appraisal, 49
 care of equipment 198-201
 deterioration of equipment, 198
 distribution of anesthetic to tissues, 38
 emergence from 49
 for normal vaginal delivery 224
- Inhalation anesthesia fundamentals 36-50
 induction, 43
 problems in 44
 inhaled concentration in 36
 maintenance of 48
 practical application, 43
 preparation of patient 43
 pulmonary phase 36
 pulmonary uptake in 38
 respiratory obstruction during 44
 storage of equipment, 200
 technique 43
 theoretic considerations 36
- Inhalation therapy, 239-246
 adjunctive, 243
 anesthesia and, 246
 carbon dioxide 245
 humidified gas, 243
 increasing inspired oxygen 240
- Injuries to eyes, 168
 to lungs 169
- Instruction in anesthesia 250-254
 interns, 253
 medical students 250
 residents, anesthesia, 254
 surgical 253
 tracheal intubation 75
- Instructions to outpatients, 220
 to parents 219
- Intensive therapy, unit, 191
- Intern instruction in anesthesia 253
- Internist, choice of anesthesia by 8
- Interview preanesthetic 3
- Intravenous barbiturate anesthesia, 84-94
 appraisal, 93
 clinical use 89
 complications 91
 apnea 93
 arterial hypotension 93
 coughing and laryngospasm, 92
 extravascular injection 91 173
 intra arterial injection 92, 173
 thrombophlebitis 92
 vomiting 93
 controllability, 84
 failure to block afferent impulses 86
 induction 90
 injuries due to 173
 maintenance 91
 muscle fasciculations after 93
 postanesthetic course 93
 selection of patients 89
 theoretic considerations 84

- Operating room, preparation of patient in, 18-20
- Operation anesthesia during 124-177
- Opiates, effect on circulation 155 See *Narcotics*
- Opium alkaloids, preanesthetic use, 12 See *Narcotics*
- Outpatients, anesthesia for 219-221
instructions to 220
- Oxygen
properties, 28
toxicity due to 243
- Oxygen apnea 242
- Oxygen consumption, regional blood flow and 40
- Oxygen physical properties in cylinders 27-28
- Oxygen therapy, 239
apnea due to 242
by face mask, 241
by nasopharyngeal catheter 240
by tent or hood 241
dangers of 241
dilution with helium 244
under increased pressure 245
- PAIN emergence excitement due to 195
immediate postoperative 187
treatment 193
- Palpation blood pressure measurement by 33
- Palsy brachial caused by anesthesia 171
- Paralysis vocal cord after tracheal intubation, 185
- Parasympathomimetic action of barbiturates 88
of cyclopropane 55
of thiopental 88
- Parents instructions to 219
- Partition coefficient 38
- Pediatric anesthesia 211-218 See also *Children*
- Pentobarbital preanesthetic use 10
- Pentothal See *Thiopental*
- Pentylentetrazol in narcotic poisoning 236
- Peripheral circulatory system
depression from local anesthetics 120
effect of cyclopropane 54
of ethyl ether 56
- Peripheral nerves injury during anesthesia 171
- Peroneal nerves common injury during anesthesia 172
- Phenergan See *Promethazine*
- Phenobarbital, preanesthetic use, 10
- Phenylephrine
effect on arterial hypotension, 159
in inhalation therapy, 244
in narcotic poisoning 234
in treatment of arterial hypotension, 166
- Piliso hex in sterilization of equipment, 200
- Physical status classification 6
- Picrotoxin in narcotic poisoning 236
- Pin index system on gas cylinders, 22, 27, 29
- Pitocin with cyclopropane, 228
- Planes of anesthesia 139-142
- Pneumomediastinum due to gas pressure 169
- Pneumonia postoperative, 135
- Pneumonitis, chemical 171
- Pneumothorax as hazard of operation 186
due to anesthesia 170
- Podalic version anesthesia for 227
- Poisoning narcotic management, 231-238 See also *Narcotic poisoning*
- Pontocaine See *Tetracaine*
- Poor risk, 5
- Pop-off valves 25
- Position of patient changes during transportation hypotension and 165-181
relation to arterial hypotension 165
- Postoperative period 180-201
immediate, hazards 180-187
- Postural hypotension See *Hypotension postural*
- Potassium chloride in ventricular fibrillation 208
- Preanesthetic medication 9-16
appraisal 15
barbiturates 10
belladonna drugs 14
excessive hypotension due to 155
in children 211
narcotics 12
purpose of, 10
respiratory depression and 11, 12
sedatives 10
- Preanesthetic period 2-16
- Preanesthetic rounds 2-4
before spinal anesthesia 96
for children 211
- Pregnancy complications of anesthetist and 223

- Morphine**
 effect on respiration, 11
 in treatment of postoperative excitement, 196
 postoperative use, 194
 postural hypotension due to 12 13
 preanesthetic use 12 13
- Motion** effect of on anesthetized patient 165, 181
- Muscle fasciculations** intravenous barbiturates and, 93
- Muscle relaxants** 65-71
 action, 65
 antagonism to 68
 appraisal 71
 dosages 66
 effect on circulatory system, 71
 on respiration 69
 metabolism and excretion 67
 postoperative respiratory depression from, 186
 synergism with 69
- Myelitis**, transverse after spinal anesthesia 113
- Myocardial infarction** during anesthesia 165
- NALLINE.** See *Nalorphine*
- Nalorphine** in infant resuscitation 229
 in narcotic poisoning 237
- Narcotic poisoning**
 analeptic drugs in 236
 appraisal 238
 central nervous system depression in 235
 diagnosis, 234
 electroencephalogram in 235
 gastric lavage in 235
 management 231-238
 prognosis, 238
 treatment, 231 235
- Narcotics** as cause of asphyxia neonatorum 229
 in early stages of labor 223
 postoperative use 194
 preanesthetic use 12
 dosage 13
 sensitivity to 14
 side actions 12
- Nasopharyngeal catheter** for oxygen therapy, 240
- Nasotracheal intubation**, 78
- National Fire Protection Association** recommendations for safety, 150
- Nebulization** of cardiovascular stimulants 244
- Nembutal.** See *Pentobarbital*
- Neostigmine**, antagonism to muscle relaxants 68
- Neo-synephrine** See *Phenylephrine*
- Nerve block** pudendal for obstetric anesthesia 227
- Nerve fibers** electrical activity, 118
 respiration of, local anesthetics and 119
- Nerves**, injury during anesthesia 171
- Nervous system** See *Central nervous system*
- Neurologic sequelae** after spinal anesthesia 113
- Neurophysiologic basis** of anesthesia 42
- Nikethamide** in treatment of postoperative excitement 197
- Nisental** See *Alphaprodine*
- Nitrous oxide**
 anesthetic use 55
 diffusion anoxia due to 186
 in early stages of labor, 224
 in normal vaginal delivery 224
 partition coefficient 38
 properties 27 28
- Norepinephrine** in narcotic poisoning 234
 in treatment of arterial hypotension 166
 liberation in ether anesthesia 56
- Novocain** See *Procaine*
- Nupercaine** See *Dibucaine*
- OBSTETRIC analgesia and amnesia** 223
- Obstetric anesthesia** 222-230
 cesarean section 227
 complications of 225
 continuous caudal block 226
 early phases of labor 223
 for normal vaginal delivery 224
 for podalic version 227
- Obstruction** respiratory during inhalation anesthesia 44
- Ocular problems** after spinal anesthesia 112
- OEM mask** 241
- Open drop anesthesia**
 appraisal 64
 errors in 64
 induction 62
 maintenance 64
 preparation 61

- Secobarbital preanesthetic use, 10
 Seconal See *Secobarbital*
 Secretions accumulation during inhalation anesthesia, 47
 thick, treatment of, 244
 Sedatives preanesthetic use, 10
 Sensitivity to local anesthetics, 121
 to narcotics, 14
 Shock anesthesia for patients in, 41
 Signs of anesthesia 143
 Sodium lactate in ventricular fibrillation 207 208
 Sources of ignition, 151
 Spinal anesthesia, 95-115
 abducens palsy following, 112
 agents and techniques 96
 ancillary preparations, 101
 circulatory effects, 159
 delayed sequelae, 107
 dosages, 97
 equipment 100
 sterilization, 101
 for cesarean section, 227
 for normal vaginal delivery, 225
 headache after, 107
 hearing difficulties after 111
 hypobaric, 99
 neurologic sequelae, 113
 ocular problems after, 112
 period following injection 105
 preanesthetic approach 96
 pre existing neurologic disease and 114
 serial injection 99
 specific gravity of solution 98
 technique injection, 105
 lumbar puncture 102
 use of vasopressor drug 98, 159
 vs epidural anesthesia 114
 Spinal anesthetics See *Anesthetics*
 spinal
 Stages of anesthesia 138-142
 Static sparks fire hazard of 152
 Status physical classification 6
 Sterilization of equipment for inhalation anesthesia 198
 by chemicals 199
 by heat 200
 for spinal anesthesia 101
 Storage of anesthetic equipment 151 200
 of cylinders 151
 Strap restraining 190
 Student medical instruction in anesthesia 250
 Succinylcholine
 action 66 67
 dosage, 66
 in treatment of laryngospasm 185
 metabolism and excretion, 67
 Surgeon anesthetist relationships 175-177
 Surgical manipulation, arterial hypotension due to, 162
 Surgical resident, instruction in anesthesia, 253
 Suralta See *Barbiturates, intravenous*
 Synchron See *Decamethonium*
 Systole manual, 206 See also *Cardiac arrest*
 TABLE, operating positioning of patient on, 18, 19
 Tears, artificial 169
 Techniques
 closed system, and explosion 151
 inhalational, 43 130
 intravenous 90
 local, 122
 spinal 102
 tracheal intubation, 74
 Temperature regulation during anesthesia in children 215
 Tensilon, antagonism to muscle relaxants 68
 Tetracaine, spinal, dosage 97
 duration of action 97
 Thiomytal See *Barbiturates intravenous*
 Thiopental
 complications due to 91
 effect on respiration 87
 equilibration of 85
 fate in body 85
 metabolism 85
 rectal use in children 215
 stages of anesthesia with 139
 technique of administration 90
 See also *Intravenous barbiturate anesthesia*
 Thorazine See *Chlorpromazine*
 Thrombophlebitis due to intravenous barbiturates 92
 Tilt, head up as test of circulatory competence 13 15 162
 Trachea intubation 72-83 See also *Intubation tracheal*
 Transfusion blood in children 216
 reactions to 162 183
 use in hemorrhage 162
 Transtracheal injection 78

- Preoperative measures, immediate, 18-20
- Preparation
 of children for operation, 211
 of outpatients for operation, 219
 of patient 18
 for inhalation anesthesia, 43
 for intravenous barbiturate anesthesia 89
 for open drop anesthesia 61
- Pressor drugs See *Vasopressor drugs*
- Pressure, blood See *Blood pressure*
- Pressure gauges, 22
- Procaine as local anesthetic, 117
 in extravasation of thiopental 92
 in treatment of cardiac arrhythmias 120
 spinal dosage 97
 duration of action 97
- Promethazine
 effect on circulation 15 156
 on respiration, 11, 15
 preanesthetic use, 14
- Propylene glycol in inhalation therapy, 244
- Pudendal nerve block for normal vaginal delivery 226
- Pulmonary blood flow, inhalational anesthetics and 37
- Pulmonary edema, oxygen in treatment of 245
- Pulmonary interstitial emphysema, 169
- Pulmonary phase inhalation anesthesia 36
- Pulmonary uptake inhalational anesthetics 38
- Pulmonary ventilation during anesthesia in children 215
 in overdose of anesthetic 51 158
- QUELICIN See *Succinylcholine*
- RACHI RESISTANCE, 106
- Radial nerve, injury during anesthesia 172
- Reactions transfusion 162 183
- Records
 anesthetic, 124-136
 example of 125
 follow up notes 136
 instructions for filling out 125
 hospital, preanesthetic notes for 4
 recovery room 190
- Recovery room, 188-192
 benefits of 188
 design and facilities, 189
 intensive therapy unit 191
 personnel of, 188
 records, 190
 release from, 191
- Rectal anesthesia 215
- Reflex traction, 163
- Relaxants, muscle See *Muscle relaxants*
- Reserpine effect on course of anesthesia 157
- Reservoir bag 23
- Resident anesthetic instruction, 253
- Surgical instruction in anesthesia 253
- Respiration
 controlled apnea due to, 70
 depression during induction of inhalation anesthesia, 44
 effect of cyclopropane 52 53
 of thiopental 87, 88
 effect of muscle relaxants, 69
 of preanesthetic drugs on, 11 12
 of promethazine 15
 in children 213
 obstruction, immediate postoperative 183
 paralysis during spinal anesthesia 106
- Respiratory sequelae immediate postoperative 183
- Respiratory tract notes for anesthetic chart, 135
- Responsibilities of surgeon 175
- Restlessness postoperative, 195
- Restraining strap 190
- Resuscitation cardiac, 204-210
 anesthetist's role 208
 tracheal, 185
- Resuscitator Kreiselman, 232
 pulmonary 245
- Reticular activating system, 42, 43
- Risk, surgical 5
- Rounds preanesthetic, 2-4
- SAFETY measures in prevention of fire and explosion 151
 in use of gases 27
- Scoline See *Succinylcholine*
- Scopolamine preanesthetic use 14
 relation to postoperative excitement, 195
- Secobarbital effect on respiration 11

- Trichlorethylene, 58
 in early stages of labor, 224
 Trifluorethyl vinyl ether, 59
 Trilene See *Trichlorethylene*
 Trimar See *Trichlorethylene*
 Trimethylene See *Cyclopropane*
 Tuberculosis patients, antiseptic precautions with, 201
 Tubes, endotracheal See *Endotracheal tubes* and *Intubation, tracheal*
d Tubocurarine
 action, 66
 dosage, 66
 metabolism and excretion 67
 synergistic action with ether, 69
 See also *Muscle relaxants*
- ULCERATION, ether, 169
 Ulnar nerves injury during anesthesia, 172
- VALMID See *Fthiminate*
 Valves pop-off, 25
 regulating, 30
 Vaporization bottles 24
 Vasopressor drugs
 dosages 167
 effect on arterial hypotension 159
 use in spinal anesthesia 98
 in treatment of arterial hypotension, 166
- Vasoxyl See *Methoxamine*
 Venipuncture, traumatic, 173
 Venous pressure, cyclopropane and 54
 Ventilation, pulmonary, during anesthesia in children, 215
 establishment in narcotic poisoning 231
 Ventricular fibrillation, treatment, 208
 Vinethene See *Ether divinyl*
 Vision difficulties with after spinal anesthesia, 112
 Vixit preanesthetic 2
 Vocal cord paralysis after tracheal intubation, 185
 Vomiting
 due to intravenous barbiturates 93
 during inhalation anesthesia, 47
 from narcotics, 12
 immediate postoperative, 184
- WESCODYNE cleaning of equipment by 199
- XENON as anesthetic 59
 Xylocaine See *Lidocaine*
- ZEPHIRAN in sterilization of equipment, 200

